RECORD OF MEDICINE

GENERAL PRACTICE CLINICS

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PHILOSOPHICAL PERSPECTIVES OF MOTION SICKNESS — *Félix Martí-Ibáñez*

SYMPOSIUM ON MOTION SICKNESS. PART I.
Guest Editor—MAJOR HERMAN I. CHINN, USAF

EDITORIAL — *Herman I. Chinn*
LABYRINTHINE FACTORS IN MOTION SICKNESS — *Leslie N. Gay*
THE NONLABYRINTHINE CAUSES OF MOTION SICKNESS — *Robert S. Schwab*
HEAD MOVEMENTS AND MOTION SICKNESS — *W. H. Johnson*
CENTRAL NERVOUS PATHWAYS OF EXPERIMENTAL MOTION SICKNESS — *S. C. Wang and Robert L. Tyson*
THE SELECTION OF MOTION SICKNESS-SUSCEPTIBLE INDIVIDUALS — *A. A. J. van Egmond, J. J. Groen, and G. de Wit*
PASSENGER COMFORT IN COMMERCIAL AIR TRAVEL WITH REFERENCE TO MOTION SICKNESS — *Ludwig G. Lederer and George J. Kidera*

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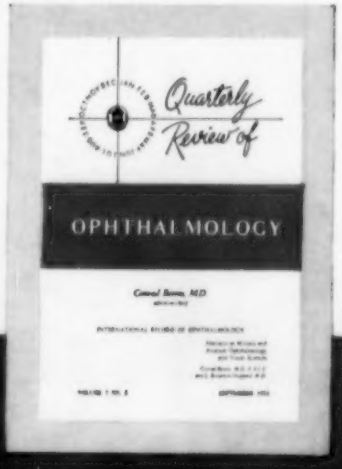

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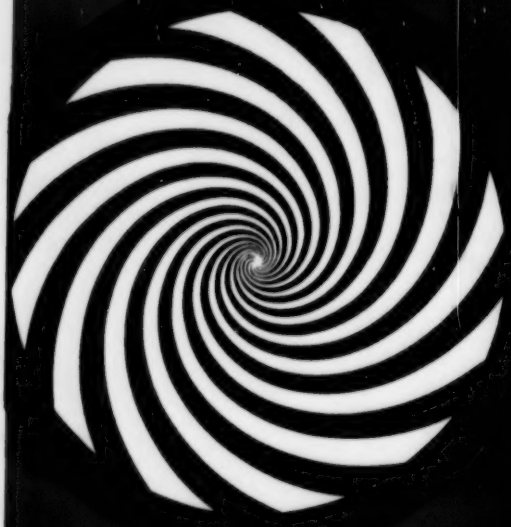

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* Part II of the **Symposium on Motion Sickness** will be published in the *International Record of Medicine*, January, 1955. The following articles will be presented in Part II:

MEDICATION FOR MOTION SICKNESS PRIOR TO AND DURING WORLD WAR II
by R. L. Noble, M.D., Ph.D., D.Sc., F.R.S.C.

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† A biographical note on Major Herman I. Chinn will appear in the January, 1955, issue of *International Record of Medicine*.

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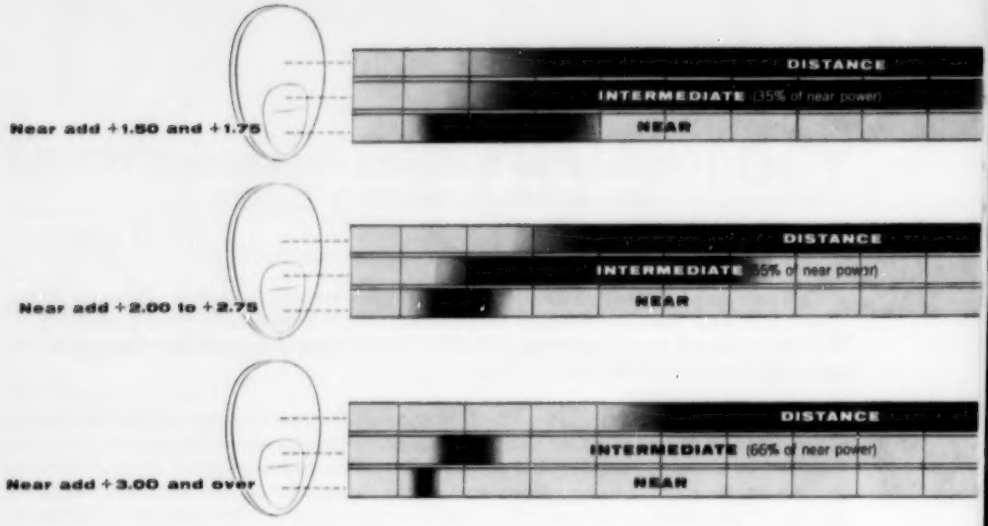

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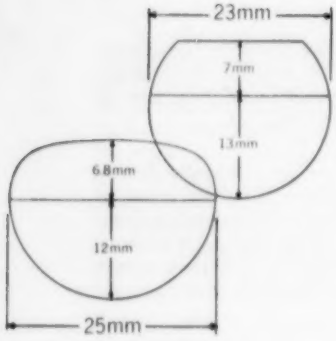

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Philosophical Perspectives of Motion Sickness

Félix Martí-Ibáñez, M.D.

NEW YORK, N. Y.

For thousands of years man lived a "two-dimensional" life in a tridimensional world. While man used his legs, horses, or carriages as his principal means of transportation, the physical displacement of his body involved a number of difficulties and discomforts which, however, rarely affected his mental and organic well-being. But when man abandoned *terra firma* and took to the water, when he furrowed rivers, lakes and oceans in rafts, canoes and boats, he had to pay the penalty exacted by Nature: seasickness.

Nausea and vomiting were associated for thousands of years with seafaring and only occasionally with land journeys. Seasickness overtook Ulysses and his companions in the Homeric saga; it was a scourge to the Spanish *Conquistadores* when their proud boats cut into boundless seas under virginal stars; it laid low the Portuguese heroes who first circumnavigated the globe; and it continues to vex those who, through necessity or pleasure, plough the seas in modern transatlantic liners.

Since literature and history relate so few instances of motion sickness overtaking horsemen—no matter how violent, fast, or exhausting their travels—we cannot subscribe to the ancient theory that nausea is caused by mere visceral displacement. It is possible, however, that the men who rode on the backs of the lurching elephants that were responsible for Pyrrhus' costly victories suffered from motion sickness. The men, astride the camels—ships of the desert—led by Colonel T. H. Lawrence during his famous rebellion in Arabia Deserta, told of the nausea which overtook them on the hot, singing sands that resembled an infinitely monotonous gray sea.

At the beginning of our century, man decided to give up the dimensions in which he had moved horizontally for several millenia and to attempt a vertical conquest of space. The extraordinary motility of airplanes added new violence to motion sickness and made this condition a tremendous problem in an age when entire countries, like some in South America, have made a sudden transition in their transportation from mule to plane. It was then that medical research workers began to investigate motion sickness not as a vexatious if inevitable companion of the travelling

mortal, but as a medical riddle whose physiopathology was a question mark, while its therapy had been compounded of folklore and crude empiricism.

The progress made by civil and military aviation has emphasized the importance of studying and preventing motion sickness, a problem that naturally embraces sickness produced by airplanes, boats, automobiles, trolley cars, and other means of physical displacement of human beings. The immense progress made in our grasp of the physiopathology and therapy of motion sickness is illustrated by this current Symposium, organized and directed by Major Herman I. Chinn, an international authority in this field, who, along with a brilliant group of research workers, has described the panorama of motion sickness in a truly impressive group of papers. This vast mural of motion sickness will be depicted in this and the next issue of the INTERNATIONAL RECORD OF MEDICINE, and it represents the first international monograph ever published on this subject.

The historical and literary references to *mal de mer* are as ancient as they are numerous. Cicero, the golden-tongued Roman statesman, said that he "would rather be killed than again suffer the tortures of *nausea maris*." Before him, Hippocrates pointed out that when a hellebore draught was administered to a patient to provoke emesis, the patient "should move about and sleep and rest as little as possible." This advice was based on his concept that "sailing the seas shows that movement upsets the body."

Throughout the history of Medicine we find frequent references to seasickness as well as attempts to interpret it medically. William Hyde Wollaston (1766–1827), the eminent ophthalmologist famous for his research on the physiological mechanism of central vision, tried to show that seasickness was caused by "oscillatory alteration of the blood," especially in the brain, brought about by the movements of the ship.

The notion of seasickness suddenly widened with the expansion of the semantic cage in which the elusive and fluttering problem was imprisoned. In 1881, J. A. Irwin used the term "motion sickness" for the first time; he felt that seasickness was a misnomer, for this condition occurred not only on lakes and rivers, but could "be induced by various other motions than that of turbulent waters." His term was universally accepted only at the beginning of the Second World War, when the unforgettable Sir Frederick Bantig stated that the expression "motion sickness" could include a series of syndromes resembling seasickness but resulting from "frequently repeated oscillatory movements of the body."*

Finding a name for an illness is often tantamount to focussing light on possible treatment. This has occurred time and again in the history of Medicine: we need only recall the therapeutic implications in the great Renaissance physician and poet Fracastoro's coining of the name "syphilis." At any rate, as the semantic concept of classical seasickness changed to "motion sickness," a conceptual revolution was effectuated: the idea that the sea caused nausea was replaced by the theory that *movement* was responsible for such a disturbance, a concept pregnant with new therapeutic possibilities.

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* E. Horowitz, "Motion Sickness"—The East African Medical Journal, Vol. 31, No. 1, Jan. 1954. Pages 13–21.

Next, the ideological scope of the word "nausea" was extended. Since *nausea* was derived from *naus*, the Greek word for vessel, it at first meant only seasickness, but eventually came to mean an urge to vomit arising from other causes. In his recent novel *La Nausée*, Jean-Paul Sartre used the term existentially to signify the physiological expression of a supreme philosophical disgust with life—an idea whose physiological roots certainly bear a strange resemblance to the anxiety factor in motion sickness.

The study of motion sickness, or kinetosis, was parallel to that of certain reactions observed in the human being when he undergoes changes of position, e.g., the change from the supine to the erect position in the orthostatic, or orthotic, reaction. Among the different types of kinetosis, investigators began to include physiological reactions that were provoked by oscillations passively determined on the body, either in a swing or in a boat, by rhythmic or irregular movements, angular, linear, radial, circular, or seesaw accelerations.

Clinical facts accumulated quickly. It was observed that motion sickness ran the gamut of pallor, cold sweat, difficulty in breathing, weak pulse, nausea, vomiting, and physical and moral prostration. Certain sensory, visual, tactile, and olfactory excitations produced a similar picture, as did acute tobacco intoxication. The neurovegetative system actively participated in motion sickness, and there was a marked analogy between this syndrome and the phenomenon of vagal excitation produced by physostigmine. Paradoxically, visceral shaking-up was not enough to excite the nerve endings in the abdominal viscera, since ordinary shaking provoked by racing and jumping did not produce motion sickness. Notice was also taken of the absence of motion sickness in deaf-mutes and of the similarity between motion sickness and Menière's syndrome. Proof was adduced that odors, fear, and anxiety aggravated motion sickness, or provoked it in certain cases. Motion sickness of long duration, during extended sea voyages when the weather is constantly stormy, could cause arterial hypertension, loss of weight, dehydration, exhaustion with acidosis, and mental depression.

This avalanche of observations made in laboratories and clinics created a doctrinal corpus around motion sickness. But this corpus was an amorphous one, awaiting the patient hand that would transform the chaos into cosmos, give organic structure to the huge pyramid of empirical facts, and train a clear light down from the experimental apex of that pyramid to its wide clinical base.

The most revolutionary step taken in the study of the factors causing motion sickness was the decision to study motion sickness independently of its most obvious organic localization, the stomach; it was decided to seek its genesis in the recondite areas of the organs governing equilibrium. Today it appears logical that motion sickness should be studied in connection with the organs responsible for equilibrium, but years ago it seemed only a remote possibility, since the predominant tendency in the historical evolution of Medicine has always been to begin the study of an illness in the area of its principal symptom. Only in our time has Medicine begun to evolve philosophically from outside in, from the symptom to the etiology, from the dramatic picture of the manifestations of an illness to a subtle grasp of its causes.

With admirable skill Dr. Chinn and the other authors of this Symposium study

motion sickness. In this brief introduction, which is like the diffuse tuning-up preceding the rising of the concert-curtain, we are concerned solely with pointing out some of the philosophical aspects of the research into the causes, prevention, and treatment of motion sickness. Such work is of the utmost importance if we bear in mind that more than 25 million people travel by plane every year, and that plans for interplanetary navigation are taking more concrete shape all the time.

The philosophical concept of motion sickness can be summed up by saying that this condition is caused by changes in the spatial stability of the environment in which the human being lives. Man exists on a planet to which he is bound by a great number of factors. He derives his sustenance from the crust of the earth; he is born on earth and to earth he returns at death: Earth is man's dwelling place. Psychoanalysis—especially the Jungian school—has shown how deeply the archetype of Mother Earth is imbedded in man's unconscious mind. It is of little consequence that the new Physics and modern Astronomy have taught man that earth is not his true cosmic center. Psychologically, earth has been man's focus from the time he first appeared on it, and the emotional realization that the earth is the center, axis, and basis of his existence is more important to man than the intellectual concept that our planet is not the center of the universe. The earth is as much a symbol of stability for the human being as his knowing where he stands at each moment. Maps teach man his position on the planet; guide books give him his location in a city. This visual orientation and intellectual recognition of his geographical placement tell man *where* he is. And to know *where* one is, is, to a certain degree, to know *why* one is there. To know one's location on earth at all times is an indispensable requisite for peace of mind.

Man is a creature who is mentally everywhere in the universe, but physically he is bound to one specific spot on earth at a certain time. All changes in the relation of man to space affect the very roots of his life. Man has adapted himself to his environment and has arranged his life basing it on distance and space. Ironically, technology has abolished distances and thrown man into the vertiginous whirlpool of an age where, in order to conquer Space, man has destroyed his own philosophical and physical stability. Motion sickness is a penalty for man's spatial restlessness.

The physical theory of man's position on earth is physiologically located in three sensory organs: the vestibular apparatus, sight organs, and the proprioceptive system. The orientation, coordination, and regulation of all these organs are indispensable in the maintenance of equilibrium. As De Wit has stated,* the sense of equilibrium is connected with the inertial system of the earth. The principal function of the vestibular apparatus is to register at all times the position of the body on earth. Equilibrium in the human being is maintained by means of the collaboration of the vestibular organ, proprioceptive system, and muscular system.

Any number of examples would bear out these basic principles. A person without labyrinths would drown if thrown into the water. Skaters gliding through a thick mist over the frozen surface of a lake would trace circles indefinitely if they lost their

* Seasickness, G. De Wit, Acta oto-laryng. Suppl. CVIII, 1953. Stockholm. Page 11.

spatial orientation. A dove without labyrinths cannot fly; and if its eyes are bandaged, it will return to earth.

Motion sickness occurs when there are disturbances of the vestibular function along with kinesthetic and psychological alterations. Irritation of the vestibular apparatus produces nausea and vomiting, and if that apparatus is destroyed, it will bring about total immunity against swing sickness. Such disorders are also brought about through disturbances of visual orientation, when the horizon is seen to be moving, or when positional orientation is changed in a plane, or by the rolls, pitches, yaws, and scends of a ship. The common denominator of all motion sickness is the exposal of the human being to angular, linear, or radial accelerations.

According to Christopher Shaw, the dynamic cause of motion sickness is the same on earth as it is on water or in the air, it being the sum of linear accelerations and decelerations according to longitudinal, lateral, and vertical axes and around them, especially so-called linear accelerations of pitch, roll and heave and, to a lesser extent, of yaw, surge, and sway.

Apparently, the higher species of animals all suffer from nausea under the same conditions that produce nausea in human beings, as De Witt has observed in monkeys, dogs, seals, and parrots. On the other hand, persons with altered vestibular function brought about, for example, by an extended streptomycine treatment, causing auditive nerve lesions, are immune to motion sickness.

The initial psychological symptom of motion sickness is disorientation with respect to our spatial position on earth. Optical illusions, such as the feeling that things around us are moving, or that the horizon is shifting, contribute to an increase of motion sickness. Psychological disorientation creates a state of psychic discomfort followed by a sensation of uncertainty. The proprioceptive system then sends an avalanche of abnormal alarm stimuli to the brain to warn it that the body is occupying an abnormal position in space. These alarm stimuli—flash signals warning us of impending danger—cause a dis-inhibition of the mesencephalic center and bring about symptoms of alarm, thereby aggravating motion sickness. Then when we become conscious of sick people around us, the exogenous psychic factor—psychological contamination—may intervene. The victim of motion sickness, threatened by the disturbances of his equilibrium, becomes aware of a cosmic threat to the stability of his Ego. This important aspect of the motion sickness threat, which reaches into the depths of one's psychological structure, is one of the philosophical roots of motion sickness still to be explored.

The therapeutic and prophylactic revolution in motion sickness began with one of those "lucky" accidents so frequent in Medical History. In 1947 Gay and Carliner used dimenhydrinate at the Johns Hopkins Hospital to treat a female patient suffering from hives, and accidentally cured her of car and trolley sickness which had plagued her all her life. Every time the patient took dimenhydrinate for her hives, she could travel by automobile or trolley without difficulty. Later, on November 27, 1948, on board the 13,000-ton ship "General Ballou," sailing from New York to Bremerhaven with 1,300 soldiers, "Operation Nausea" took place. Dimenhydrinate was then successfully used during a frightful storm, the test being made with controls. Since then that drug has become part of the therapeutic arsenal against seasickness, to which have been

added several other drugs. Anti-motion sickness agents, like Bonamine, are successfully used today against pernicious vomiting during pregnancy (the disease that killed Charlotte Brontë!),* against vomiting caused by radiation therapy, Menière's syndrome, fenestration interventions, and cerebral arteriosclerosis.

The history of motion sickness is as ancient as man, for at first it followed his horizontal displacements on earth, and later his vertical ascent through endless space. Yet, until quite recently, no work had been done on it. A new horizon in physiology, pharmacology, and prophylaxis is open to research workers. This Symposium, so ably directed by Dr. Chinn, offers a vast picture of the extent of our progress in this matter. But the symbolic centaur, Chiron, still gallops through the plains of Medical History. And only the future years can tell what new and promising therapeutic results research workers will derive from their present investigation of *nausea maris*. Their success will enable man, already master of the seas, to proceed ahead with the conquest of outer space.

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* Marriott, H. J. L.: Medical Milestones, Baltimore, Williams & Wilkins, 1952. Page 270.

SYMPOSIUM ON MOTION SICKNESS

Editorial

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Motion sickness is a by-product of man's inventive genius. It was born when the first susceptible individual ventured forth in his primitive craft on turbulent seas. As trains, automobiles, and airplanes were developed and vehicular transportation became universal, opportunities for experiencing this condition increased exponentially. Today, there are relatively few adults who have never suffered some degree of discomfort resulting from motion. There are, of course, a few hearty individuals who are invigorated by turbulent weather. At the other extreme are those chronic sufferers who never complete a journey without distress. The vast majority, however, cover a broad spectrum between these extremes. It has been shown that with moderately rough seas about 30 to 50 per cent of all persons will become sick and, under violent conditions, virtually everyone will succumb.

As with any widely experienced malady, the causes and treatments were glibly propounded by physicians and laity alike. The older medical journals are storehouses of ingenious theories and fanciful remedies. Fortunately, neither motion sickness itself nor the measures adopted for its alleviation were permanently disabling, so that each individual could blissfully continue his own special concoction or maneuver. Controlled experiments were virtually unknown. With the onset of World War II, the necessity for delivering large numbers of troops in optimum fighting condition necessitated an abrupt change from this leisurely policy. Intensive studies were initiated not only to determine the most effective medications, but also to learn more of the basic physiology involved. Workers in this country, in Canada, and in England were especially active. It can be safely stated that the few war years placed the treatment of motion sickness on a sounder basis than the decades, or even centuries, preceding this period.

With the close of the war and its immediate practical problems, interest in motion sickness waned. It was spectacularly revived in 1949 with the demonstration that the antihistamine dimenhydrinate was a markedly effective prophylactic. This report stimulated extensive screening of new drugs until, today, there is an embarrassing wealth of effective preparations. Over a dozen preparations are now available, capable of protecting half or more of otherwise susceptible individuals. Each drug has its loyal advocates and enthusiastic manufacturer's support.

In addition to these developments, there has been renewed interest in the basic physiology underlying motion sickness. Significant advances have been made in elucidating the afferent pathway of the emetic stimuli, in the role of head movement in motion sickness, in the selection of susceptible subjects, and in other related areas. Approximately half of the following articles were selected to discuss the causative factors of motion sickness, and the remaining half its prevention or treatment.

Labyrinthine Factors in Motion Sickness

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Motion sickness may be described as a physical state that develops in human beings and animals when they are subjected to oscillatory movements over which they have no control. This definition implies that the term should be applied to those symptoms that develop when the victim is detached from the earth as in a car, plane, or ship.

Relatively recent studies of the labyrinth have elaborated the earlier classic experiments of Flourens[1] and have crystallized the conclusion of most observers that the labyrinth is the most important anatomic area in the causation of motion sickness. The vestibular or labyrinthine structures consist of auditory and nonauditory portions. The auditory portion consists of the cochlea, and perhaps the saccule. The nonauditory portion consists of the semicircular canals and the utricle. Each of these canals is paired and forms part of the inner ear. These canals are to be found in the petrous part of the temporal bone.

The bony labyrinth consists of a series of channels closely approximating the contained soft tissue structures known as the membranous labyrinth, and these channels are separated from the bony walls by a fluid, the perilymph. The membranous labyrinthine structures are filled with a similar fluid, the endolymph.

The semicircular canals consist of two vertical and one external canal to each set. Each of the canals is curved, each has an expanded portion known as the ampulla, and communicates at its extremities with the utricle. Nonexpanded portions of the vertical canals join to communicate with the utricle through a common orifice.

The receptor organ of the semicircular canals lies in the ampulla of each canal and is called the crista acustica. Surmounting it is the cupula, a dome-shaped structure consisting of sensory hairs, which are free to move in any direction with the flow of endolymph. The detailed anatomy and physiology of the utricle, the cochlea, and the saccule can be found in the texts of Best and Taylor[2] and of Fulton.[3]

It should be emphasized that the primary function of the semicircular canals is the registering of changes in the rate of motion acceleration or deceleration and in the direction of motion. Various technics to stimulate the labyrinthine structures have yielded much information; for example, rotation of the body, caloric stimulation through syringing the ear with hot or cold water, and galvanic stimulation.

In order to discover possible causes of motion sickness symptoms and the physiologic site of such disturbances, investigators attempted to reproduce the symptoms by artificial means. Tyler and Bard,[4] having carefully combed the literature that pertains to the various studies, reviewed these investigations.

Noble and his colleagues[5] studied the effect of three components of a swing (vertical, horizontal, and angular) on susceptible dogs. He reported that the composite motion was found to be more effective than any one of its components. McIntyre,[6] using human subjects, confirmed the impression that repetition of a combination of accelerations in different planes constitutes a more potent stimulus than repetition of any

one acceleration. Wendt and his colleagues,[7,8] using a "wave machine," examined the time intervals between accelerations. Their investigations were prompted by the observation that movements that cause large accelerations with short pauses are rarely nauseating, whereas those that cause small accelerations with long pauses tend to cause nausea and vomiting. The results confirmed these findings. The investigators concluded that the total energy per wave is a more potent factor than is the interval between accelerations.

For many years visual factors have been suspected as causes of motion sickness. That vision sometimes plays a role is shown by the fact that some pilots of aircraft are susceptible to motion sickness when they travel as passengers. When they pilot the plane, they direct their eyes toward the horizon and experience no discomfort from the motion. Proper orientation of vision assists in preventing motion sickness.

The effect of body position on the production of motion sickness was studied by several early investigators, as well as by investigators during World War II. Irwin[9] was the first to suggest that in a horizontal posture, which alters the position of the head and consequently the position of the labyrinth, a subject is less disturbed by motion than in an upright position. Tyler[10, 11] reported a sickness incidence of 30 per cent among troops required to crouch in small landing craft as compared with an incidence of 11 per cent among troops allowed to stand upright. He believes that "exclusion of visual orientation as a result of crouching was doubtless one factor." Furthermore, he believes that the fixation of the head and of the labyrinth caused by holding a crouched position for some time is a factor favorable to motion sickness.

The role played by the cerebral cortex was studied by Bard,[12] who reported no significant change in the susceptibility of decerebrated dogs. A study of the electroencephalograms of subjects gave no evidence that deviations from the normal suggest either greater or less susceptibility to motion sickness.

A number of "bodily disturbances mechanically evoked by motion" have been considered as causes of motion sickness; for example, visceral displacement, the distribution of blood in the large vessels and in the organs of the body, and the tonus and motility of the stomach. All these, as well as others, have been given consideration. Tyler and Bard,[4] having analyzed the experimental studies relative to the gastric alteration during various forms of vestibular stimulation, summarize with the statement that although such stimulation "tends to reduce or abolish the tonus and motility of the stomach, the available experimental evidence does not justify the conclusion that these gastric alterations are in any way causative of the nausea and emesis brought on by exposure to motion."

One of the important factors that has been assumed to play a major role in motion sickness is the psychologic factor. Well controlled psychologic studies give little evidence that individuals having psychosomatic complaints or psychiatric emotional instability have either greater or less susceptibility to motion. Certain investigators suggested that fear and tension are responsible for airsickness, but results of controlled studies indicate no psychologic factors of importance in the etiology of motion sickness.

Scientific evidence has established the knowledge that any disturbance of the labyrinth by trauma or by disease might induce nausea, vomiting, or dizziness. There is presumptive evidence that a minor disturbance of this organ produced by the motion

of a ship, a land vehicle, or airplane results in the same symptoms. The work of the author[13] on the prophylactic and therapeutic control of vestibular disturbances with dimenhydrinate emphasizes this evidence.

Following the original observation by Gay and Carliner[14] that dimenhydrinate prevents motion sickness in the majority of susceptible travelers, the author treated 63 patients suffering from acute or chronic labyrinthine symptoms. Their ages ranged from 16 to 74 years. The average dose of dimenhydrinate was 50 to 100 mg. every four hours; the majority of the patients took it for at least seven months.

The 63 patients were separated into three groups. In group I were the 6 patients seen within three weeks after the onset of dizziness, nausea, and vomiting. In group II were 41 patients who had suffered from dizziness, nausea, and vomiting for periods varying from 2 months to 25 years. All these patients were either cured completely or had only infrequent and minor attacks as long as they continued to take dimenhydrinate. In group III were 16 patients with symptoms similar to those in group II; all failed to respond to dimenhydrinate. In fact, in 5 patients the dizziness became more pronounced after 100 mg. of the drug had been taken. The dizziness, nausea, and vomiting were so severe in 4 of the patients that the vestibular nerve was sectioned intracranially with no relief. Dimenhydrinate, administered in doses of 400 to 600 mg. a day, failed to give the slightest relief.

In other words, 74.6 per cent of the 63 patients with inbalance were greatly improved after taking the drug. Many of the patients had to continue maintenance doses to remain free from dizziness. This demonstrates the possible site of the action of such drugs as dimenhydrinate prescribed to those susceptible to motion sickness.

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The Nonlabyrinthine Causes of Motion Sickness

*Robert S. Schwab, M.D.**

INTRODUCTION

There are four contributing causes of motion sickness[1, 2] in addition to the disturbances from labyrinthine stimulation, which have been discussed elsewhere in this symposium.

The first and second of these deal with sensory stimuli—smell and vision. They are essentially contributing causes and are frequently ineffective if labyrinthine sensitivity is absent. The third is psychogenic and includes all sorts of anticipatory feelings dealing with the various aspects of travel, as well as true conditioning from previous experiences of being ill from motion. A fourth, or miscellaneous group, includes increased susceptibility in females during menstruation, aggravation in patients with peptic or duodenal ulcer, and a vague group precipitated by overindulgence of either food or drink prior to travel. In the fourth group should be included the few subjects with such generalized disease as sinusitis, hypertensive vertigo, migraine headaches, who otherwise might weather the exposure to motion without onset of nausea and vomiting.

CLINICAL SIGNS AND INCIDENCE

Before discussing the etiologic factors of motion sickness it is well to review the symptoms commonly encountered.[3] Motion sickness is often defined for statistical evaluation as that point in the syndrome where nausea and vomiting ensue. This definition is useful for statistical purposes in the evaluation of preventive or treatment measures. The syndrome, however, involves a large number of minor symptoms that build up before actual nausea and vomiting occur. The first symptom is rather a subjective one and is described as an uneasy feeling with a certain amount of lack of interest in the task being done, the book being read, or the person with whom one is talking. No visible signs are shown by the subject at this point and a great many travelers bothered by motion sickness may pass through this phase alone and never develop further symptoms or complaints because of the termination of their trip. Such passengers would not admit to being ill even though they were aware of this rather subtle change in their normal habits.

With more motion this mild lack of interest in the immediate environment increases steadily and is accompanied by a certain amount of yawning. At this point there may or may not be visible changes in the person's skin color, which may show as pallor, and a keen observer might note both the yawning and the lack of interest as prodromal to motion sickness. At this time anorexia develops, as well as social indifference.

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As the syndrome goes further, evidence of autonomic disturbances increases. Salivation that the subject is aware of is usually accompanied by perspiration over the forehead. A subjective feeling of coldness and a desire to shiver may occur and, at this point, efforts to close the eyes and drift off to sleep may be tried without success. From now on, as the symptoms progress, subjective feelings of depression, isolation, and anxiety may occur. There is usually complete apathy if any kind of motor activity is suggested and the subject is convinced that he is going to be ill very shortly. Then symptoms of nausea and vomiting may occur precipitously. It is to be noted that the four contributing factors mentioned in the first paragraph are most likely to produce nausea and vomiting if they are encountered during this prodromal build-up of the syndrome.

The dynamics that cause the nausea and vomiting in motion sickness were investigated during World War II at two centers studying motion sickness by means of barium fluoroscopic studies. Both Campbell[5] and Goehring[6] found that when nausea and vomiting were induced by means of a swing, a definite series of changes appeared in the barium fluoroscopic examination of the stomach after the swinging had stopped. These were: (1) an increase in secretion so that the stomach became rapidly full; (2) a disappearance of the normal gastric motility; (3) a very definite spasm of the pylorus, which effectively closed off the normal exit of the stomach.

As these three findings developed in the subject, they were accompanied first by nausea and then by active vomiting. In addition, in some 30 per cent of subjects who had been ill with motion sickness for a long time, there was a suggestion of increased rugal markings that are seen in chronic gastritis. Subsequent investigation of these patients by means of the gastroscope by Benedict[7] showed that the mucosa was entirely normal and there was no gastritis.

It is apparent to anyone working with this subject that there is a division among humans of susceptibility to motion sickness. In the author's work in the naval service during the late war, about half of the subjects could be classified under an arbitrary division as Type I and the other half under Type II.[4] Type I is defined as people who have a constitutional susceptibility to motion of any kind. They invariably give histories of swing sickness, car sickness, or train sickness going back as far as they can remember. Even very moderate or very slight motion has precipitated serious and severe forms of motion sickness. There seems to be no natural tendency on their part to develop tolerance when the motion is continued, as on a long voyage, and they remain ill until the voyage stops. Weight loss during this period is a conspicuous feature. In slang terms, they are the proverbial "landlubbers." Approximately 1 to 2 per cent of the general population fit into this category. Type II represents the remainder of people who become seasick if the motion is rough enough but who very frequently survive moderate voyages involving motion without being ill. They tend frequently to show only the prodromal symptoms on short trips. They are more prone to develop tolerance as the voyage continues ("sea legs"). Weight loss is usually absent. They do not give a history of previous motion sickness on swings, cars, etc. It is hard to be sure of any information about the incidence of this Type II in the general population, since nearly

everyone could be made ill under the proper circumstances. Somewhere around 30 per cent of the normal population group might fit into Type II.

PHYSIOLOGY

The mechanism of the induction of motion sickness by the four causes mentioned above is not too difficult to understand if we realize that the motor site of both increased salivation and vomiting lies in the vagal nuclei in the medulla. These vital parasympathetic centers lie across the main paths of incoming sensory stimuli from eye kinesthetic sensation (median longitudinal bundle) and pain sensation of the descending root of the fifth nerve. They also are close to the hypothalamic centers that tie in with that vast network of the rhinencephalon that is involved with taste, smell, and many emotional states. The memory of visceral experiences, such as previous nausea from motion, is believed to be a function of the temporal lobe and rhinencephalic systems, both of which are intimately associated with these autonomic motor centers that produce vomiting.

There is a hint, too, that the reticular activating nerve cells in the midbrain, which are so important to maintaining the normal alert state, are involved in the syndrome of motion sickness. This would account for early signs such as yawning, drowsiness, and general lack of interest before nausea is experienced. Motion sickness, therefore, is essentially a diffuse syndrome as far as localization in areas of the central nervous system is concerned, particularly when it is fully developed with symptoms of hypotonia, bradycardia, excessive perspiration, small pupils, somnolence, loss of motor coordination, loss of motor skills, and a conspicuous group of higher psychic functions such as indifference, seclusiveness, depression, and apathy. All of this confirms the importance of the vagus centers, which can interfere with normal motor behavior and mental equilibrium in such striking ways.

EXAMPLES

The various types of nonlabyrinthian-induced motion sickness will be described by giving clinical examples of each. The *first* to be considered is the sensation of smell.

Smell

It has long been known by travelers that they are more uncomfortable in rough weather in crowded, stuffy places where unpleasant smells dominate. To avoid this on shipboard one is advised to stay out in the open air as much as possible. It is not easy to prove that specific olfactory stimuli, unless they are associated with previous episodes of seasickness, contribute in all cases to bringing on the symptom complex. It is not very scientific to describe the interior of a cabin of a ship as being "stuffy" without specific knowledge of the gaseous elements involved in this expression. There is no evidence that increase in carbon dioxide, which conceivably might occur in a crowded stateroom, has any bad effects that induce nausea. What is probably meant is the presence of body odors, and inevitably the occurrence of an odor of vomiting, which becomes strongly conditioned with previous episodes of

sickness, even those not induced by motion. The negligible amount of engine fumes in cabins are not considered specific causes for motion sickness. The combination of unpleasant odors and feelings of being crowded in a small space probably is the best explanation for the belief that the fresh air on deck is a preventative of seasickness.

For example, on a rough sail on the author's boat his sister, who is subject to motion sickness, was able to prevent it as long as she sat in the cockpit. On going below in the cabin for a brief period to put on a coat, she experienced an immediate attack of nausea and vomiting, attributed to the smell of oil and bilge water, plus musty and wet mattresses and blankets. Two other persons who were not suffering from motion sickness were unable to detect any of these odors when they went below.

Another example occurred on an airplane with a passenger who had been sick on some previous flights and not on others. During this particular one the passenger felt well since the motion of the plane was not too violent. Seated behind this passenger was a more susceptible individual who asked the hostess for a paper bag and promptly vomited. The smell of this drifted across the seat whereupon the passenger first mentioned immediately became ill and remained so until the plane landed one-half hour later. Here the smell of vomitus, possibly complicated by the mental image of the patient vomiting and the sounds produced by the act, acted as a powerful combined stimulus to bring on the symptoms, although the degree of motion was minimal.

Vision

Various forms of visual stimuli are described in the literature as contributing to the onset of motion sickness. Close use of the eyes, as in reading small print, should be avoided by susceptible persons in rough weather. It is better to keep the visual attention on distant objects, such as the horizon, rather than to watch rapid movements, such as the rail of the ship in relation to the sea. Some advise susceptible individuals to close their eyes and reduce the stimuli on the visual system as much as possible. There is not good agreement on the best procedure to follow.

For example, when a medical officer on an LST in the Pacific in the last war, I was watching a member of the port 40-millimeter anti-aircraft battery, whom I knew was susceptible to motion sickness. He was standing his watch to the left of the plane spotter who was using 7 by 50 binoculars. There was a moderate swell and very little roll or pitch, since the vessel was heavily laden. The subject was perfectly well until his turn came to look for planes through the binoculars. This complicated coordination of the eye muscles with the addition of an enlargement of the distant field produced uneasiness after approximately five minutes, then nausea. Finally, 20 minutes after the onset of the task, the sailor was violently ill, with both nausea and vomiting.

Another example is the account of a naval medical officer who recently told the author of the visual effect of artificial motion. This physician was very susceptible to seasickness and had been ill a number of times in his military experience in the late war. He attended the theatre in New York, where the stage was made to roll and move like a ship, and where the actors simulated this by changing their positions at each roll. After a few minutes of watching this artificial motion, a feeling of ma-

laise came over the subject; he had a headache over his eyes and increased salivation in his mouth—a well-known warning that he was going to be ill. His wife noted his pallor and the onset of large drops of perspiration on his forehead. He became violently ill with nausea and vomiting: he experienced no direct motion whatever and no other stimuli, except the visual one plus the various conditioning effects that were brought out.

A third example was reported to me by a sentry on an army transport who was susceptible to motion sickness but was doing well at his post, having taken a capsule of army preventive medicine that morning. He began to watch the rotating sweep of the radar scanner on the foremast and a few minutes later felt nausea and dizziness. Fortunately, he was relieved at this time and was able to go to his bunk and lie down. He was not actively sick. Subsequently he avoided watching any rotating object when he was on duty, or when it was rough and prevented further symptoms.

Psychic

The third group deals with a rather indefinite number of psychiatric factors or emotional causes. Many well-integrated and healthy personalities who are susceptible to motion sickness vigorously reject the notion that motion sickness is related to emotional instability. We have previously described, in the Navy, a group of individuals defined as Type I who were constitutionally susceptible to any kind of motion. In 51 subjects, or 74 per cent, there was a definite history of neurotic traits, and 57 per cent showed a history of fainting spells and dizzy spells not associated with motion. In the Type II group, defined as susceptible to motion sickness in rough weather, the incidence of neurotic traits was only 24 per cent, and 14 per cent showed a history of fainting or dizzy spells. There were 55 subjects studied in this group.

Many of the older articles on seasickness refer to cases where a susceptible or nervous individual could go to bed on the ship, while it was tied to the dock, and become very ill in a few hours, even though the ship had not left, simply from the anxiety related to anticipation of the motion. On the other hand, there have been well-documented instances of patients who have been sick from motion and recovered promptly under a stimulus associated with danger, combat, or increased responsibility and interest.

For example, a sailor on the transport Pollock had been ill nearly two days in November, 1942. He recovered promptly when the ship crashed into the rocks off Newfoundland and was in immediate danger of breaking up. Another example of this occurred on a yacht that was being sailed through a tricky group of shoals. A member of the crew had been ill nearly an hour in the gentle swell and promptly recovered after a bump or two on a sandbar, and the assignment to this person of the sounding lead for the next hour of sailing.

A previous unpleasant experience with motion sickness produces a most complicated conditioning in the susceptible individual. Part of the sickness produced in the case of the officer under visual stimuli might be explained on this conditioning phenomenon. In 1942 at a naval hospital, I had under my care a naval lieutenant

who was tremendously susceptible to motion sickness and who had had a trying and upsetting month on a cruiser in the North Atlantic. The patient had been ill the entire time, with a weight loss of 30 pounds, a classic Type I case. He recovered promptly in the hospital setting and, although recommended by the medical staff for a shore assignment, he was ordered to report to the *Normandie*, which was then being fitted out as a transport, and obviously a vessel of sufficient size to make motion sickness less likely in this individual. When given his orders by the Commanding Officer of the hospital in the latter's office this individual promptly vomited all over the floor and was ill for nearly two hours, precipitated entirely by the dread and reluctance of going to sea again even on a large ship.

The second individual, a physician with a strong susceptibility to motion sickness who had suffered for two months in the North Atlantic, was a voluntary experimental subject to see if artificial habituation could be accelerated by incremental periods on a swing. The first day he was able to stand the swing for four minutes before he was made ill (a person surviving for 15 minutes without nausea was considered free of the susceptibility). The second day, instead of increasing his resistance to the motion, he was made sick in two minutes and 30 seconds. On the third day, as he entered the corridor in the naval hospital where the swing was attached, he became nauseated and vomited promptly at the sight of the apparatus. Further efforts of positive conditioning toward habituation were promptly abandoned in this patient.

Miscellaneous

The fourth group of contributing factors to motion sickness include sensitivity in females at the time of their menstrual periods, and increased sensitivity of those who have partial but minimal pyloric obstruction due to duodenal ulcer.

An example of the first group was a nurse in the army medical corps, who successfully crossed the Atlantic on a small transport during rough weather without being ill, but who became nauseated and vomited in the quieter waters of the Mediterranean when her menstrual period began.

The second example concerns a retired surgeon who reported that he was most susceptible to all kinds of motion up to the age of 35, when he had a duodenal ulcer resected and a posterior gastroenterostomy installed. For the rest of his life, up to the present (75 years old), he has never had any motion sickness of any kind even in very rough weather on small vessels. It has been suggested from this experience, and others similar to it, that in a critical situation where a susceptible person feels sufficiently motivated to correct it, he submit to this type of surgery to his stomach.

In this same group, as mentioned previously, there are individuals who are made worse in their susceptibility by various forms of overindulgence prior to travel. An engineer reported that he deliberately would overindulge in smoking, drinking, and eating the night before a trip. This would produce, on the following day, when he was on a ship at sea, a violent and protracted "hangover," frequently with nausea and vomiting—regardless of the weather being rough or smooth. This was usually followed by improvement and insensitivity to motion sickness. If he did not do this he was certain, he said, to become ill from the motion of the ship and remain ill for

a considerably longer period of time than if he deliberately induced a hangover as described.

In general, however, it is considered essential to avoid such overindulgence to ensure normal gastrointestinal functioning as motion begins with rough weather.

CONCLUSIONS

In summary, there are a number of contributing causes to motion sickness outside of the labyrinth. These are: (1) olfactory; (2) visual; (3) psychic or emotional, including conditioning of various types; (4) miscellaneous, consisting of menstruation, duodenal ulcer, and the aggravating effects of overindulgence before travel.

Since all of these may play an important precipitating role in motion sickness, which may then last for a period of time incapacitating the individual in his travel, the awareness of these causes should be considered in all plans for the prevention and treatment of motion sickness.

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Head Movements and Motion Sickness*

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Although it is apparent that different physiologic and psychologic factors are important in the etiology of motion sickness, recent experimental evidence has established that the nonauditory membranous labyrinth in the head is an extremely important sensory receptor in this regard. Evidence for this comes from the fact that surgical removal of the organ in experimental animals results in immunity to this malady; this was first done with dogs[1, 2] and later with cats.[3] In regard to human beings, it is significant to note that subjects with eighth-nerve deafness have been reported as being immune to motion sickness.[4] Furthermore, it has been noted by several investigators that an acute vertigo often associated with marked motion sickness follows movement of the head in one plane of space when the body is already rotating in another plane. Voyachek[5] states that in 1908 he carried on such experiments with a centrifuge and reported that if in the course of a uniform rotation one has to change the position of the head or the whole body, as for instance turning the head through an angle of 90 degrees, the endolymph of the semicircular canals will acquire fresh impulse to shift as a result of new forces thereby produced. These results have also been noted by several investigators when using a human centrifuge. They found that during movement of the head at right angles to the plane of rotation of the rest of the body, a completely false sensation of rotation resulted. This sensation was increased the more rapidly the head was turned or the body rotated, and was often associated with an acute motion sickness. Spiegel[6] also has noted the effectiveness of simultaneous angular motions in space to induce sickness when using a Barany chair.

Controlled laboratory experiments were recently carried out by Johnson *et al.*,[7] when it was demonstrated that individual differences in susceptibility to motion sickness are to a large extent dependent upon concomitant movements of the subject's head in response to movements imposed on the body as a whole. Preliminary recordings of head movements showed that they may be complex even though the subject is exposed to such a uniform motion as that of the simple swing, and a highly significant correlation was obtained between the over-all concomitant head movement and the incidence of motion sickness.‡

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‡ The head movements of blindfolded subjects on the swing were first recorded mechanically, using a linen thread passing through a pulley connected from the subject's head to a pen which recorded the movements of the head on a kymograph. The mechanical recording was then replaced by a more accurate electronic device. This was accomplished by surrounding the subject's head with a homogeneous electromagnetic field, which enabled recording of head movements on a galvanometer by induced currents in coils attached to the head.

The importance of these independent head movements as an index of motion sickness was emphasized when they were prevented. Of 27 subjects who had vomited when the head was free to move, 24 showed no symptoms of motion sickness under similar conditions on the swing when their head was fixed by means of a strap passing around the forehead from the back of the seat. Other similar tests were carried out in regard to head fixation by using 200 subjects. A group of 100 controls with their heads free to move showed a sickness incidence of 37 per cent, and another 100 subjects with the head fixed to the back of the seat showed only a 5 per cent incidence of sickness. Similar prevention of motion sickness, using dogs as experimental animals, has been produced in these laboratories.

A practical application of these findings in the prevention of airsickness was then carried out.[8] Over 500 airborne troops were deliberately exposed to violent air turbulence. Head movement was restricted by aluminum headrests installed in the aircraft to adapt to variations in individual head size. In order to eliminate psychologic influences (and provide controls), every other subject was given a placebo in the form of a lactose pill instead of a headrest. The results of this extensive field trial established that the headrest devices had a consistent effect of preventing air motion sickness under all turbulence conditions, and that they were significantly effective under very rough conditions. A statistical analysis of the results indicates that immobilization of the head in aircraft would reduce airsickness by 60 per cent to 83 per cent, depending on the degree of turbulence involved.

Other experiments have been carried out by these laboratories in an attempt to determine the relative role of various types of head motion in producing nausea. A new laboratory device has been devised and is proving much more effective than the swing as a controlled means of inducing motion sickness in the laboratory (Fig. 1).

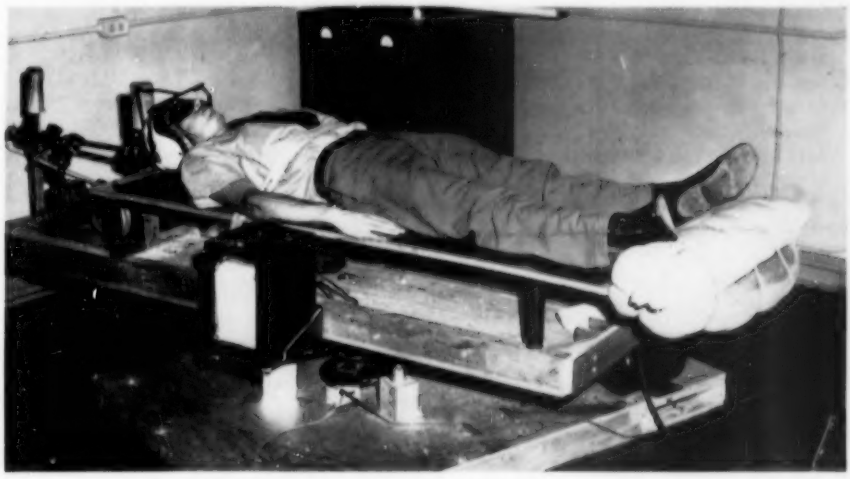

FIG. 1. Turntable with head movement activator adjusted for simultaneous head movements with horizontal rotation of the rest of the body about a vertical axis.

The body is strapped to a rotating platform in the supine position. Forced head movements are added during horizontal rotation of the body, and can be made either coronal or sagittal. The arc of head motion and timing are under variable control. Furthermore, the speed of the turntable can be altered. Controlling the above types of movement, incidence of sickness can be varied up to a maximum of 95 per cent within five minutes of exposure in an unselected group. In some cases sickness has been induced within 15 seconds.

The general significance of these findings in regard to head movement is applicable to all forms of motion sickness—seasickness, airsickness, car sickness, etc. Restriction of head movement is a therapeutic measure in the prevention of all forms of motion sickness. This may be accomplished either with or without the aid of special headrests. In vehicles with a high-backed seat, sufficient restriction of head movement to overcome motion sickness is often gained simply by holding the head against the back of the seat. However, when this is not possible, restriction of head movement may be done voluntarily; passengers can be warned to move the head slowly and only when necessary.

The use of drugs may also prove a valuable adjunct to head restriction in the prevention of motion sickness. However, research still remains to be done on the effectiveness of such combined therapeutic measures.

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Central Nervous Pathways of Experimental Motion Sickness*

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That the central nervous system is involved in the ancient problem of motion sickness was appreciated as early as 1881 by Irwin,[1] who initiated the term now applied to the series of events that results from the stimulus of motion and that terminates in vomiting. On the basis of the physiology known to him, Irwin implicated the cerebellum and the optic lobes in his discussion of the nonauditory labyrinth and its role as the seat of pathology in motion sickness.

Much has been learned since Irwin's time about the anatomy of central pathways of possible importance to this topic. It was the revival of interest in motion sickness in the past decade, however, that made possible the emergence of an over-all view of functional importance. Many studies have been purely pharmacologic and have provided valuable and provocative information. Unfortunately, the present primitive status of our knowledge about the central site or sites of action of most pharmacologic agents precludes the precise delineation of the central processes involved in motion sickness. On the other hand, stimulation and ablation experiments in animals have not only demonstrated the importance of certain central structures in the physiology of motion sickness and the relative unimportance of others, but they have also elucidated what may be termed "the final common pathway of vomiting," the medullary vomiting center with its afferent and efferent systems.[2–5]

This discussion will concern itself primarily with those developments that have contributed to our knowledge of the central mechanism of motion sickness since the comprehensive review of the subject of 1949 by Tyler and Bard.[6]

THE VOMITING CENTER

The several centers described in the brain stem have as a common denominator, by definition, the concern of execution of certain coordinated responses that involve various muscle groups and afferent routes. "Physiologically, the center is a device to establish connections, which is dependent upon the peripheral situation, and whose organization is directed toward a definite result."[7] Anatomically, each center is a group of neurons interconnected in various ways and, although it may not be differentiated by histologic technics or topographically well circumscribed,

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its limits may be defined by stimulation or by destruction. The concept of a vomiting center is older than the term "motion sickness" by 16 years, for in 1865, Giannuzzi postulated such a structure.[8] Thumas in 1891,[9] by destruction of a midline area in the posterior rhomboid fossa, and Hatcher and Weiss in 1923,[10] by destruction of the sensory nuclei of the vagi, demonstrated the existence of a vomiting center at a different locus. In 1949, Borison and Wang[11] were able to locate the vomiting center by electrical stimulation with the aid of the Horsely-Clarke stereotaxic instrument in decerebrate cats. Vomiting was elicited in these animals, as determined by expulsion of stomach contents, by stimulating the nucleus and fasciculus solitarius and dorsolateral border of the lateral reticular formation (fig. 1). Subsequently, data from destruction experiments in dogs were presented by Wang and Borison,[2, 12] in which the animal became refractory to intravenously injected apomorphine and intragastrically administered copper sulfate. Lesions were made deep in the medulla by electrocautery[2] and by radon seed implantation.[2, 12, 13] Histologic sections after demonstration of the emetic refractory state showed the vomiting center to be in the dorsolateral reticular formation, as did the stimulation experiments.

AFFERENTS TO THE VOMITING CENTER

The weight of available evidence suggests a representation of the vomiting center as in figure 1, in which the coordination mechanism for emesis is activated on reception of impulses generated by the stimulation of certain receptor sites, and in which the vomiting center in no case functions as a primary receptor.

A. The Vagi and Sympathetics

The afferent pathways concerned in the vomiting of experimental animals to intragastric mustard,[14] experimental peritonitis,[15, 16] and pyloric distention[17] have been studied. The vagi were found to be of primary importance by some,[14, 15, 17] and others[16] reported the necessity of sectioning both vagi and splanchnics to prevent vomiting. In man, there are many reports[18] that vagotomy does not interfere with vomiting, and Okawachi[19] states that in rabbits "vomiting"* induced by continual vertical acceleration is not prevented by bilateral vagotomy. The emetic action of copper sulfate in dogs was studied in 1951 by Wang and Borison.[20] It was shown that in small doses intragastric copper sulfate stimulated receptors, presumably in the gastric mucosa, which sent impulses craniad along the vagi and, for some distance at least, along the sympathetic trunks (fig. 1). The vagi appeared to carry the majority of impulses, but sympathectomy as well as vagotomy were required to produce a refractory animal. Intravenous copper or larger oral doses of copper, in animals with denervated gut, acted to stimulate emesis by a mechanism to be discussed subsequently.

One investigator has raised the problem of these peripheral pathways in relation

* Contraction of the pylorus and relaxation of other parts of the stomach were observed and interpreted to indicate that vomiting response had occurred. However, it is generally believed that herbivorous animals, such as rabbits, do not ordinarily vomit.[18]

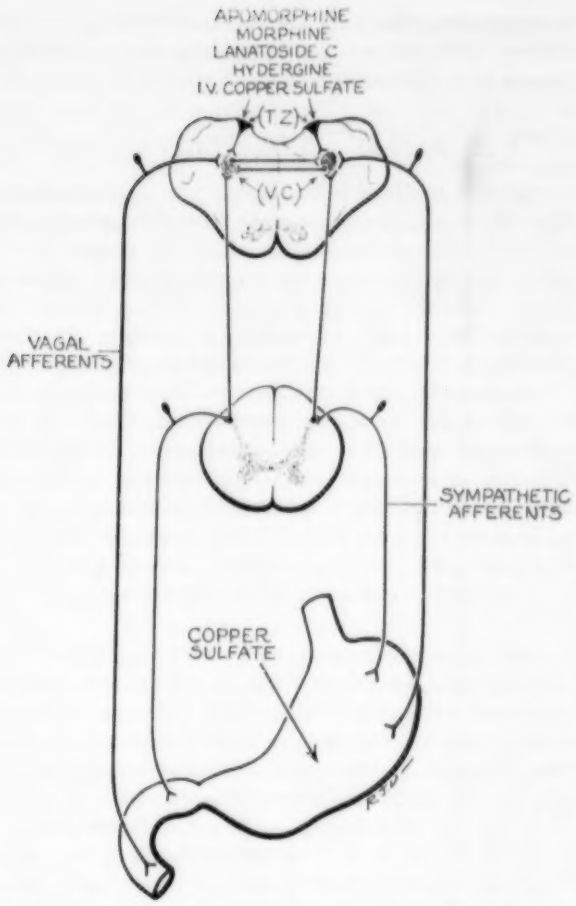

FIG. 1. Schematic representation of afferent pathways for vomiting elicited by "central" and "peripheral" emetic agents (redrawn from Wang and Borison.[4]) TZ—chemoceptive emetic trigger zone; VC—vomiting center. Apomorphine, morphine, and Hydergine act only on the trigger zone, whereas copper sulfate and lanatoside C act both peripherally and centrally.

to motion sickness. Okawachi[19] reported that rabbits, subjected to continually applied vertical acceleration, did not "vomit" after either bilateral cervical sympathetic ganglionectomy or bilateral section of the splanchnic nerves. Since there is no evidence to indicate the existence of an afferent role in motion sickness for the cervical sympathetic ganglions or the splanchnic nerves, it is difficult to harmonize these results with the fact that dogs will vomit to apomorphine or to large intragastric doses of copper sulfate after complete gut denervation (vagotomy, lower thoracic and abdominal sympathectomy[20]). To settle the problem it would seem worth while to subject animals, previously shown to be motion susceptible, to

thoracic and abdominal sympathectomy and vagotomy. Subsequent determination of their susceptibility, after recovery, would demonstrate conclusively the role of these visceral afferents in motion sickness.

B. The Chemoceptive Emetic Trigger Zone

Other than a suggestion by Hatcher and Weiss,[10] the first experimental evidence that a so-called central emetic did not act on the vomiting center itself was reported by Koppanyi in 1930.[21] Intragastric copper sulfate was able to elicit emesis in 2 dogs with chronic lesions in the dorsal vagal nuclei, whereas the response to apomorphine was irregular and the threshold appeared to be increased. This was also an indication that the dorsal vagal nuclei do not constitute the vomiting center. More recently, Wang and Borison in 1950[2] confirmed the results by ablation experiments in dogs. Subsequently, the region sensitive to apomorphine was shown[2–4] to occupy a portion of the area postrema, a narrow zone bordering on the lateral wall of the fourth ventricle, at the level of calamus scriptorius. It is separated from the more medial ala cinerea, or trigonum vagi, by the funiculus separans. This chemoceptive trigger zone, in addition, was found to be the site of action of cardiac glycosides,[3, 4, 22, 23] intravenous copper sulfate,[20] and morphine and Hydergine.[24, 25] Furthermore, dogs subjected to a bilateral ablation of the trigger zone do not vomit to a large dose of total body irradiation, which evokes vomiting in 100 per cent of control dogs.[26]

Of more immediate interest is the evidence by Wang and Chinn that ablation of the trigger zone in dogs previously found to be susceptible to the motion of a swing prevents the appearance of motion sickness.[27–29] These results strongly suggest that visceral afferents play no basic role in motion sickness. They also provoke speculation about the nature of the agents directly responsible for stimulation of the trigger zone. Brizzee and Neal[30] have recently published detailed observations on the cellular morphology of this area in the cat, following other studies with the same orientation.[31] On the basis of these observations, it is possible that either a hormonal or a nervous (axonal) stimulus, or both, is responsible for activation of the trigger zone. Thus, Brizzee and Neal[30] have described two chief cellular components. There is a group of glialoid cells resembling astroblasts in form, with one or more prominent vascular podia. These cells often appear in clusters. There is also a group of cells that these authors identify as small neurons, variable in form, larger than the glialoid cells, and possessing slender processes, which were traced only for short distances. The area is rather vascular, and loose bundles of unmyelinated fibers are seen through all parts. Of interest is the fact that very fine fibers, difficult to trace, appear to terminate on or near the cells of the area, "but are not as conclusively demonstrated as are the bouton terminaux on other regions of the medulla."[30] These authors suggest that the glialoid cells are the primary chemoceptive elements, but it appears equally possible that the fine nerve endings are the receptors in question, the cell bodies to which they lead lying in the reticular formation or in the vomiting center itself. An alternate possibility is to consider the fine nerve endings as the means by which impulses from some extramedullary

source reach the trigger zone as a relay station on the way to the vomiting center. In an attempt to answer this problem, Wang and Chinn[28] placed plastic barriers in the fourth ventricles of three dogs that had previously been demonstrated susceptible to the motion of a swing. After recovery from operation, none of the dogs became motion sick on the swing. Although suggestive of a humoral agent, further progress in this direction depends upon the demonstration of the return of susceptibility to swing sickness after removal of the plastic barrier, and also upon histologic verification of the absence of damage to the cerebellar structures.

C. Other Afferents to the Vomiting Center

It is reasonable to suspect the existence of afferent pathways to the vomiting center in addition to the chemoceptive trigger zone, the vagi, and the sympathetics.[5] That these supramedullary pathways are not of fundamental importance in motion sickness appears from the study of Bard et al.[23] with a chronically decerebrate dog. Autopsy showed that decerebration occurred in a plane from the cranial borders of the superior colliculi to about 1 mm. rostral to the origin of the third nerves. In a series of swing tests before operation, this animal was shown to be susceptible to the motion; vomiting occurred within 10 minutes. A total of 13 swing tests were made postoperatively at weekly intervals, and each time the dog vomited within nine minutes. The survival time of this animal was 145 days.

The Cerebellum. The relation of cerebellar structures to motion sickness was first investigated by Bard and collaborators,[23, 24] on the basis of the fact that the best-known suprasegmental representation of the vestibular apparatus lies in the cerebellum. A dog shown to be susceptible to the motion of a swing was decerebellated and studied over a period of 17 months, during which time it failed to vomit on any of 15 swing trials. Salivation, licking, and panting were observed during the trials. Similar results were obtained in a series of previously susceptible dogs after removal of nodulus, uvula, and pyramis, although salivation and licking occurred in the absence of panting. Susceptibility to motion was not affected by removal of pyramis and a few folia of uvula. An additional important fact determined in these experiments was the maintenance of a normal sensitivity to the emetic action of apomorphine. These results indicate that the central mechanism for the emesis of motion sickness consists of three parts: (a) the final common pathway for vomiting, including the vomiting center in the reticular formation and its motor systems; (b) the chemoceptive trigger zone in the area postrema; and (c) certain cerebellar structures, especially nodulus and uvula. The functional relationships are illustrated in simplified fashion by figure 2.

Recent investigations by Wang and Chinn[28, 25] have confirmed and extended the observations of Bard and collaborators. In table I are presented typical data from one dog in each of seven operative classifications.[25] These dogs were screened first for motion susceptibility, and only dogs that showed emesis consistently within 25 minutes of swinging on several occasions (for technics, see reference 29) were selected for surgical extirpation of parts of the cerebellum. Then the thresholds to intravenous apomorphine and intragastric copper sulfate were established. After re-

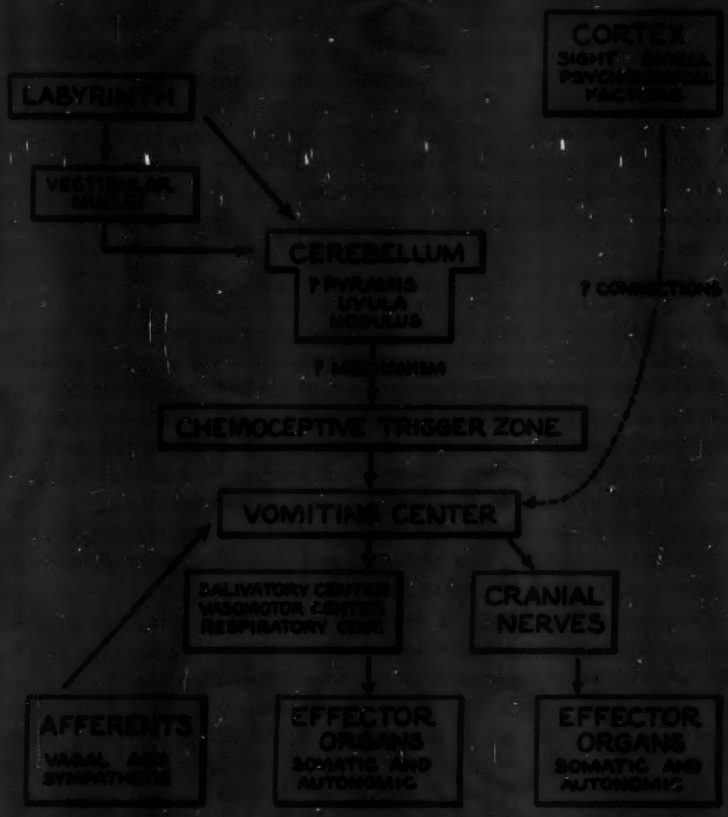

FIG. 2. Schematic representation of the central nervous pathways of motion sickness gathered from investigations by Wang and Chien.[8, 10]

operation from the operation, the chemical thresholds were redetermined and found to be not appreciably different from the controls, indicating that neither the chemoceptive trigger zone nor the vomiting center was damaged inadvertently by the surgical procedure. Swing tests were performed (testing periods up to 60 minutes), and the response was noted. The dogs were subjected to a repetition of these tests up to four to eight months postoperatively. The brains were then perfused in 10 per cent neutral formalin, sectioned and stained with cresyl violet or according to the Weil method. Examination of the sections provided information about the extent of the operative lesion. The data in table I indicate that ablation of the nodulus or uvula alone is not sufficient to prevent motion sickness. These operated dogs were found to be partially refractory to motion sickness; that is, the latency for vomiting to occur is sometimes prolonged. In order to produce a totally refractory animal, it was found necessary to remove part of uvula in addition to nodulus, or part of pyramis in addition to uvula (see table I).

The Labyrinth. Sjöberg first demonstrated experimentally the importance of the labyrinth as the primary receptor site in motion sickness,[56, 57] although he states[57] that Kreidl, in unpublished experiments, had previously rendered animals insensible to motion by bilateral section of the eighth nerves. Sjöberg's work, utilizing the technic of bilateral labyrinthectomy, has since been confirmed by a number of authors.[10, 26, 28, 51, 58] Which of the sensory organs of the nonauditory labyrinth discharge the afferent impulses necessary for the production of motion sickness is a problem beyond the scope of this discussion. Two well-known characteristics of motion sickness in which the nonauditory labyrinth and its cerebellar representation are probably determining factors are first, that people vary in their susceptibilities to a given type of motion as well as to different types, and second, that the effective motion must be applied for a minimum period of time. This latter characteristic strongly suggests a temporal summation of the afferent impulses, although the site (or sites) of summation remains to be determined.

PHARMACOLOGY

The most recent drug shown to have antiemetic potency is chlorpromazine, Thorazine, or SKF 2601-A (10-(γ-dimethylaminopropyl)-2-chlorphenothiazine hydrochloride). There are many clinical reports of its action.[40, 41] In experimental studies, chlorpromazine in dogs has afforded protection against apomorphine,[15-44] and dihydrogenated ergots.[43, 44] Since it is known that apomorphine and dihydrogenated ergots act on the chemoceptive trigger zone,[4, 24, 25] it was suggested that chlorpromazine acts by depression of this zone.[43, 44] More suggestive data[45] indicate that the chlorpromazine in small doses acts as a competitive inhibitor at the trigger zone, whereas in large doses it depresses the reticular vomiting mechanism. On the basis of these statements alone one might presume that chlorpromazine in small doses would be a potent antimotion sickness drug, since ablation of the trigger zone in dogs prevents motion sickness.[52-59] Indeed, Cook and Toner[45] have found

TABLE I

*Effect on Motion Sickness of Extirpation of Parts of Cerebellum**

Dog No.	Operation	Survival time (months)	Latency of swing tests during postoperative period†
28	Vermis (lobulus simplex, declive and tuber)	4	25, 20, 13, 9, and 16 min.
25	Pyramis	4	17, 21, 19, 12, and 14 min.
52	Nodulus	8	33, 41, 24, 54, 22, 27, 9, and 30 min.
53	Uvula	4	37, 28, and 45 min.
303	Nodulus, Uvula (partial)	4	4 times negative
203	Nodulus, Uvula	7	46 min. and subsequently 3 times negative
18	Pyramis, Uvula (partial)	4	5 times negative

* The data contained in this table are selected from Wang and Chinn.[58]

† Latency in excess of 25 min. is considered to be prolonged.

that chlorpromazine is very effective in reducing swing sickness in dogs. However, in man chlorpromazine is found to be a rather poor anti-motion sickness remedy.[47] Experimentally, chlorpromazine does not suppress the emetic action of all the drugs that so far have been shown to act on the trigger zone, such as morphine,[45] intravenous copper sulfate,[46] and lanatoside C.[44–46] It might be hypothesized that morphine, intravenous copper sulfate, and lanatoside C are bound more tightly to the receptor mechanism of the trigger zone than is chlorpromazine.[48] Thus, one cannot predict with certainty whether or not small doses of chlorpromazine will prevent emesis due to motion sickness. It would appear likely that large doses, by depression of the reticular formation, could protect susceptible individuals.

In view of the multitude of studies on the efficacy of various antihistaminics in the prophylaxis and treatment of motion sickness, it is of interest to consider the experimental evidence pertinent to their central sites of action. Dramamine (dimenhydrinate, β-dimethylaminoethylbenzohydryl ether 8-chlorotheophyllinate) and Benadryl (diphenhydramine, β-dimethylaminoethylbenzohydryl ether) have been most studied in this regard. Early studies on the effects of these compounds on apomorphine induced emesis in experimental animals indicated that some protection was afforded.[49, 50] Others[51, 52] presented evidence to show that these compounds affected the severity and frequency of vomiting but had no significant effects on its incidence. More recent papers present convincing arguments that diphenhydramine and related compounds have no effect on the vomiting elicited by apomorphine.[53–55] In view of the fact that apomorphine acts on the chemoceptive trigger zone,[4] it is probable that diphenhydramine and related compounds exert their preventive action elsewhere. Although the evidence indicates that these drugs do not protect against apomorphine (or morphine,[56] which also acts on the trigger zone), the experience with chlorpromazine as discussed above suggests that a broader screening of the antihistamines against a variety of emetics is necessary before a conclusion can be reached about the site of action.

CONCLUDING REMARKS

Experimental studies of vomiting and motion sickness should meet the following criteria: (a) a sufficient number of animals should be used to provide statistical reliability; (b) a species of animals should be chosen on the basis of reproducible responses to minimum stimuli; (c) animals should be subjected to experimental procedures only after the threshold for each animal has been determined several times for the stimulus to be tested and found to be reproducible; (d) the general health and nutritive condition of experimental animals must be maintained;[54] (e) before being subjected to the stimulus of motion or of an emetic agent, the animal must be fed. Disagreement of results can almost always be traced to the disregard of one or more of these criteria.

The simplified scheme of figure 2 summarizes the pathways discussed in this review. The experimental evidence for such a representation has been presented, and it has been used as a framework in which to view the action of antimotion sickness and antiemetic drugs.

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The Selection of Motion Sickness-Susceptible Individuals

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I. INTRODUCTION

By motion sickness we understand the feeling of malaise experienced by persons predisposed to this disease when they are exposed to changes of acceleration. There are, theoretically, two points of action of these changes of acceleration: (a) The suspension of organs or parts of organs causes them to be displaced with respect to each other or to their point of suspension. This would give rise to a stimulation of the autonomic nervous system, resulting in motion sickness. (b) The affection is caused by stimulation of the sense organs involved in the perception and maintenance of equilibrium. This is primarily the vestibular organ, in conjunction with the proprioceptive system and the eyes.

Animals susceptible to motion sickness lose this sensitivity if their labyrinths are destroyed; this makes it probable that the primary somatic cause of motion sickness is situated in the vestibular organ. In human relationships, psychic influences are, quite naturally, of paramount importance. Disorientation and lack of balance are phenomena making a deep impression on the psyche. Various investigators, however, rather remarkably attribute only a secondary influence to these psychic components; in their opinion the vestibular disturbance is the primary cause. The results of these disturbances, however, may exert an important depressive influence on the psyche (in depressive states, for example).

The vestibular apparatus is therefore probably of primary importance in the pathogenesis of motion sickness. As this feeling of malaise is in the first place the result of changes in linear acceleration acting on the subject, the point of attack should be sought for in the sense organs that mainly react to these accelerations, i.e., the otoliths. Although the relationship between the otoliths and motion sickness has been known for a long time, it is still very peculiar that, so far, little correlation has been found between the sensitivity of the labyrinth and motion sickness. This is better understood if it is realized that up to now there have been no really reliable quantitative methods of examining the labyrinth. *This correlation becomes clear, however, if we are able to use a reliable quantitative method.* Such a method is perhaps possible in future, in the form of the examination with the parallel swing. Although the results obtained in the laboratory of the otologic clinic at Utrecht are favorable, general clinical application is still difficult because of the particular technic of measuring (measurement of the blood pressure in the central retinal artery according to Baillart). It is fortunate, however, that the subject's manner of reacting to linear

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TABLE I

Schema of Examination

I	II	III	IV	V	VI
History: 1. Has patient suffered a concussion? 2. Has patient gone through a period of malnutrition?	Walking tests, and if necessary, audiogram	Cupulogram	2nd cupulogram (after 3 months at sea)	Conclusion good = not seasick, or only slightly so seasick = pathologically seasick, also after a long period	Total duration of the examination
No abnormalities	→	*a.* Normal	→	good	25 min.
	→	*b.* Too steep	*a.* Normal →	good	40 min.
		→	*b.* Too steep →	seasick (specific)	40 min.
Concussion in the history	*a.* No *marche en étoile*				
	b. Marche en étoile → (usually the audiogram also shows a mild unilateral abnormality of perception)	Dissociation between right and left	→	seasick (unspecific)	45 min.
Malnutrition in the history	→	*a.* Normal	→	good	25 min.
		b. Too steep			
		c. Irregular	→	seasick (unspecific)	25 min.

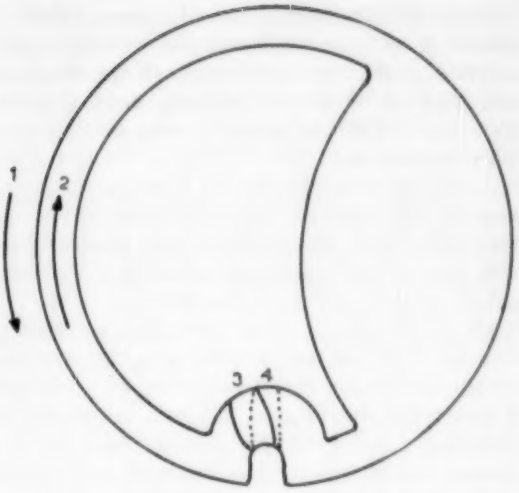

FIG. 1. Schema of the circuit formed by semicircular canal and utricle. 1—Direction of rotation of the head. 2—Direction of rotation of the endolymph-stream. 3—Position of the cupula under the influence of the endolymph stream. 4—The cupula at rest.

accelerations completely agrees with his reactions to rotatory stimuli. In other words, by investigating whether the subject shows a marked reaction to rotatory stimuli, his disposition to motion sickness can also be verified.

The rotatory stimuli are perceived by the system of the semicircular canals. In the laboratory mentioned above, a quantitative method of examination of these organs has been developed, called *cupulometry;* and the use of this method has been successful in proving the quantitative relationship between motion sickness and the labyrinth.

II. CUPULOMETRY

Cupulometry is a method of examination more adapted to the physiology and mechanism of the semicircular system than the earlier methods. This system consists of three semicircular canals all originating and terminating in the utricle. The schema represented in figure 1 holds true for each of these canals.

Each semicircular canal forms, together with the utricle, a closed ring filled with endolymph. This endolymph cannot circulate freely, but is blocked at one side by the cupula, a screen of hairs surrounded by gelatinous substance, situated on a transverse ridge, the crista. This whole apparatus is contained in the ampulla, a widening of the canal. Steinhausen was the first to prove that, *in vivo*, this cupula reaches the opposite wall and that it forms, as it were, a watertight lock-gate in the circuit (the method of proof was to inject Chinese ink into the living preparation). Various other investigators, e.g., Dohlman, observed just as Steinhausen did that the cupula is bent if an angular acceleration is given to the system. The law of inertia causes the endolymph to lag relatively behind as compared with the fixed parts, i.e., also with respect to the cupula: the latter is thus pressed aside. The cupula proves to have straightened again after some time, which means that it must be elastic. *The semicircular system therefore behaves like a pendulum.*

The formulas pertaining to such a system are all to be found in the science of mechanics: it is a torsion pendulum (Steinhausen). The mechanical problems to be solved are: Is it a slow or a rapid swing? Is the movement suppressed or not? There are still other questions from the more physiologic point of view. What is the stimulus? How does the cupula react? In what way are nystagmus and sensation correlated with the stimulus?

According to Steinhausen and Vilstrup, the cupula acts as a swinging door in the ampulla; the endolymph is subjected to the force of inertia, causing a deflection of the cupula. Even though the cupula is passive, it will try to regain its zero position, thus pushing the endolymph back slowly. A sensation of turning and nystagmus is associated with a particular deflection of the cupula. The quantitative relationships are: An impulse $\gamma°$/sec. provokes a sensation of turning round with an angular velocity of $\gamma'°$/sec. and a nystagmus, the slow phase of which shows an angular velocity of the eye of $\gamma''°$/sec.; $\gamma=\gamma'=\gamma''$. In other words, nature has developed a system that allows good judgment of the angular velocity of the head during a physiologic movement. The eyes turn back during the slow phase, in order to compensate for the movement of the head, lest the subject should lose his orientation with regard to his surroundings. The rapid phase is of central origin and is of no influence in these phenomena (Ruding).

The impulse γ is administered during the so-called clinical turning test (cupulometry). The subject is accelerated subliminally until he is turning with the desired angular velocity (e.g., 60°/sec.). If his sensations are none but those of perfect rest, the chair (turning room) is stopped suddenly (any time within 3 seconds). The process of stopping serves to administer the impulse to the semicircular canal; the endolymph tries to persist in its angular velocity while the walls of the canals have come to a standstill. The cupula is deflected to an angle of

$$\xi_0 = \gamma \frac{\theta}{\pi} \tag{1}$$

in which ξ_0 = angular deflection of cupula in degrees, γ = angular velocity in degrees/sec., θ = moment of inertia of the endolymph, π = moment of friction for unit angular velocity.

The cupula starts to move back again immediately, but, because it has to push the endolymph through the narrow canal, the time taken to return to the position of rest is rather long.

At any time t after the stopping, the deflection of the cupula is

$$\xi_t = \xi_0 e^{-\frac{\Delta}{\pi} t} \tag{2}$$

in which Δ is the directional momentum given to the endolymph by the cupula. The subject regains his sensation of rest at the moment the cupula passes the threshold of the perception of angular velocity γ_{min}. It takes a time t_u, counted from the stopping, to reach this moment:

$$t_u = \frac{\pi}{\Delta} \ln \frac{\theta \gamma}{\pi \xi_{min.}} \tag{3}$$

The essential feature is that t_u is proportional to the logarithm of the impulse, and that the rapidity of the return is principally determined by the factor π/Δ. If π (friction) is considerable, the return will be slow; if Δ (the stiffness) is considerable, the cupula will push back the endolymph more quickly. If measured under suitable conditions, the average value of π/Δ for human subjects amounts to 16 seconds. Therefore, the initial deflection of ξ_0 will be reduced to $1/e=0.37$ in 16 seconds.

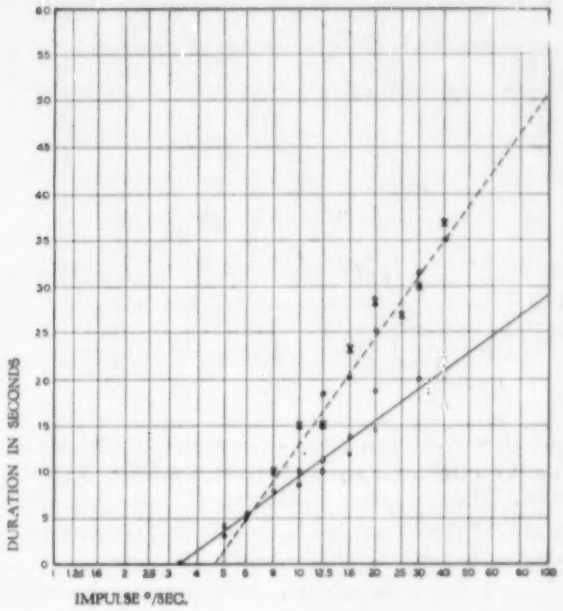

FIG. 2. Normal cupulogram. O—sensation after turning clockwise. X—sensation after turning counterclockwise. O—nystagmus after turning clockwise. X—nystagmus after turning counterclockwise.

In the study of radioactivity the rate of decay is expressed by the term "half life." When this term is applied in our case, this is 1.6 times shorter than the $1/e$ value; thus the half life of a cupula deflection is of 10 seconds' duration. The cupulogram is a graph in which the duration of the after-sensation (or nystagmus) is indicated as a function of the impulse (fig. 2). The impulse is obtained by stopping the turning room (or turning chair) suddenly (i.e., within 3 seconds) after having given it a certain velocity (e.g., 30°/sec.) by a subliminal acceleration.

It is possible to make a nystagmus cupulogram, in addition to a sensation cupulogram. The two would have to coincide or to run parallel if there were no influences exerted by central factors, and if both nystagmus and sensation cupulogram were to indicate only the function of the peripheral organ. This is, however, seldom so. The zero (threshold) values obtained in the accurate observation of the slow nystagmus phase prove to be about equal to the sensation threshold, but the nystagmus cupulogram is generally steeper and deviates less from the theoretically calculated values. The slope of the sensation cupulogram is—although characteristic for a given individual—very variable and changes from very steep to very flat. *It is exactly this*

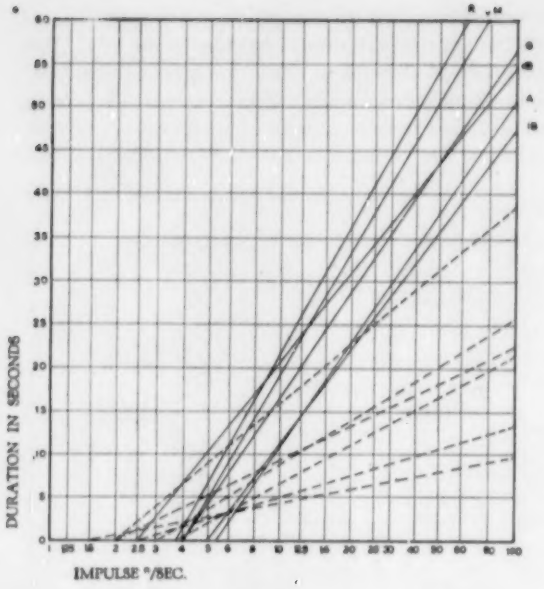

FIG. 3. Cupulograms of seasick persons, compared with those of nonseasick subjects.
- - - - - nonseasick.
——— seasick.

inclination of the cupulogram (as determined by "central factors") that may yield valuable data on the liability of a given person to seasickness. This will be shown in the following pages. The nystagmus cupulogram also shows variations in inclination, although these are not so marked as in the sensation cupulogram. We will only deal with the sensation cupulogram in this paper.

The turning apparatus must fulfill exacting demands. These requirements are: It must be free from vibration, and the subjects should not have any other sensations than those caused by the rotatory impulses administered. Hence, a turning room is better than a turning chair, because the patient can be kept completely in the dark (elimination of visual stimuli) and no current of air is felt (elimination of the tactile sense; this is why the room must be free from vibration). The process of stopping must be adequate but smooth in order to obviate undesirable sensations. In our clinic a large number of cupulograms were made, both of persons susceptible to seasickness and of individuals not liable to it. The subjects examined consisted largely of personnel of the Royal Netherlands Navy. The number of persons liable to seasickness was about 100, that of the nonsusceptible test persons about 50. *It was shown that 80 per cent of all persons susceptible to seasickness possess an abnormally steep cupulogram.*

Figure 3 shows the cupulograms of a number of seamen who suffered chronically from seasickness and of a group of seamen not liable to seasickness (all belonging to the Royal Netherlands Navy). The difference is very clear. It is a striking feature of this graph that there is no essential difference between the threshold value of seasick and nonseasick persons. The vestibular organ evidently possesses a certain adaptive

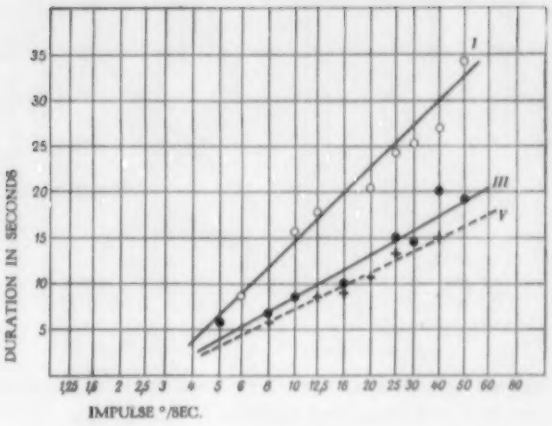

FIG. 4. Cupulograms of a test person before (I) and after 45 minutes (III) and 90 minutes' (V) rotation in the turning room.

power, which gives a permanent lowering of the curve in the long run, in which case we can better speak of habituation. *A "normal" vestibular apparatus possesses the power of adaptation, which in the long run eliminates the undesirable effects of too strong stimuli. Individuals specifically liable to seasickness lack this capacity.* This adaptation occurs very rapidly in normal cases.

Figure 4 gives a cupulogram of a nonseasick subject who had been rotated in the turning room for 90 minutes (Curve II), compared with that taken at the beginning of the test (Curve I). The boundary between persons liable to seasickness and those unsusceptible to it is at a π/Δ of about 11 seconds. All with a steeper cupulogram (i.e., with a $\pi/\Delta >$ 11 seconds) are liable to seasickness; all with a flatter cupulogram ($\pi/\Delta <$ 11 seconds) are not susceptible to it.*

We mentioned above that 80 per cent of all seasick persons show too steep a cupulogram. What is the situation with the other 20 per cent? This group proves to be divisible into two further groups:

(a) Most persons belonging to this group show, when examined very carefully, mild vestibular disturbances. On being turned to the right (in which mainly the left labyrinth is stimulated) they exhibit somewhat different values than on being rotated to the left (when the right labyrinth is stimulated). These subjects also show, in agreement with the cupulogram, a mild unilateral cochlear disturbance (deafness for high tones) and, for example, mild disturbances in walking and past-pointing tests (fig. 5).

There is almost always a concussion in the past history of these patients. In cases of

* The value π/Δ is computed in the following way for a certain cupulogram: let the duration of the nystagmus (sensation) be t_1 for an impulse γ_1 and t_2 for γ_2; take $\gamma_2 = 20\ \gamma_1$, then

$$\frac{\pi}{\Delta} = \frac{t_2 - t_1}{3}$$

This is based on the fact that $e^3 = 20$ (see formulas 2 and 3).

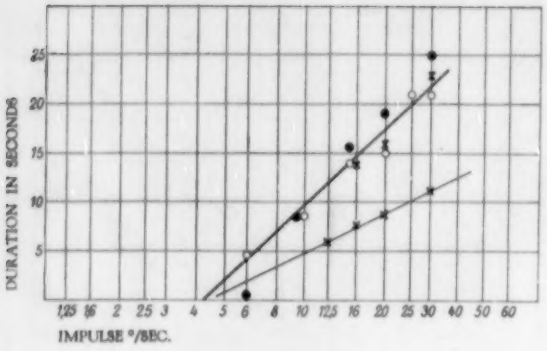

Fig. 5. Cupulogram of a non-seasick person who had suffered from concussion. The curve of the right labyrinth (O———O) shows a steeper inclination than that of the left one (X- - - -X).

chronic seasickness, it is very important to ascertain whether the patients have ever had such a cerebral trauma.

(b) In some cases the only abnormality found is a very irregular cupulogram. These people were mainly found among former prisoners-of-war who had been through a period of malnutrition. We suppose that here we are dealing with an irreversible lesion caused by vitamin B deficiency (especially of pellagra preventive factor) (fig. 6).

THE PARALLEL SWING EXAMINATION

According to many investigators, such as Arslan and Montandon, stimulation of the vestibular organ, especially with linear stimuli, causes an increase of the pressure in the central retinal artery. Hulk and Henkes found that, under these circumstances, the pressure in this blood vessel is especially increased in people liable to motion sickness. We used this labyrinthine vasovegetative reflex in our examination of seamen. We measured the pressure in the central retinal artery before and after the subject had been swung on a parallel swing for a minute.

The parallel swing used by us is a stretcher suspended on four rather long (3 meters) parallel ropes. The retinal blood pressure was measured with the ophthalmodynamometer of Baillart, which requires great skill.*

Figure 7 shows a comparison of the results in some chronically seasick individuals with those in a group of persons not susceptible to seasickness. The outcome is striking: all the persons liable to seasickness showed the autonomic reflex after rather mild stimulus, whereas among the nonseasick only one showed it. Moreover, it appeared afterwards that the latter person was a very dubious case of insusceptibility to seasickness.

The examination with the parallel swing will probably prove to be of great value for the study of seasickness. In the present state of the technic, however, too much skill is required to make it a routine method.

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* We acknowledge with thanks the assistance of our colleagues of the Ophthalmologic Clinic of the State University of Utrecht in these measurements.

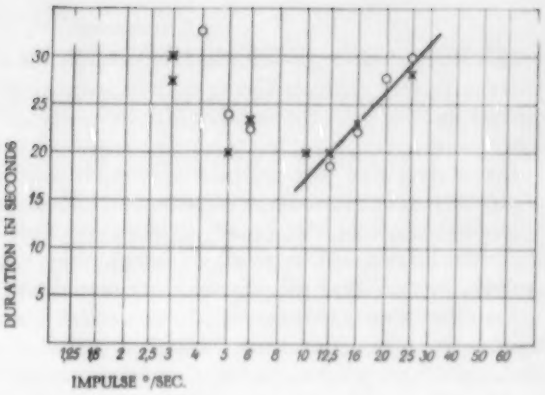

FIG. 6. Cupulogram in deficiency.

DISCUSSION

Krijger also used cupulometry in his examination of aircraft pilots. His results agree fairly well with ours, except that he found, in trained pilots, a rise of the threshold value in addition to a flat course of the curve. Aschan arrived at the same conclusion in his examination of airmen. We did not find any significant difference in threshold value, only a clear difference in steepness. Aschan also pointed to the remarkable fact that the steepness of the curve depends on the subject's being in normal condition: it takes on a steeper course when he has influenza or when he is tired. We were able to confirm this, and this objective observation is moreover in agreement with the well-known fact that a person becomes seasick more easily when in poor condition. The facts mentioned in this investigation perhaps open the possibility of tracing prospective cases of seasickness. The cupulogram is an important factor in such an examination, in addition to careful history taking (concussion, malnutrition). A steep cupulogram, which remains steep after a sea journey, might be an indication that the person concerned is permanently susceptible to seasickness. We reproduce a schema of examination, based on these ideas, as an example. It is already in practical use. The time of observation has been too short, so far, to warrant a verdict on the reliability of the results. Until now it has developed that about 2 per cent has too great a susceptibility for seasickness. This is a favorable percentage: not too many have to be disqualified.

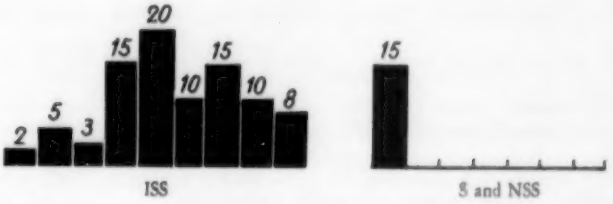

FIG. 7. Increase of pressure in Baillart units after swinging on the parallel swing shown by nine seamen liable to seasickness (group ISS) and by those not susceptible to it (group NSS); also in nonseamen (S) (total number 7).

SUMMARY

This article describes two methods of examination of the vestibular organ, which prove to be very useful in finding cases of seasickness. Cupulometry especially is a method that indicates the susceptibility to seasickness by means of a comparatively simple curve.

The investigation with the parallel swing is of great promise in this respect, but at present it is not yet suitable as a routine method.

Specific seasick persons (about 80 per cent of all cases of seasickness) show a steep cupulogram and an increased labyrinthine vasovegetative reflex, as can be observed by measuring the pressure in the central retinal artery.

The other seasick individuals (the nonspecific group) have either had a cerebral trauma (concussion) or gone through a period of malnutrition. The methods described are probably useful for recognizing persons susceptible to seasickness right at the beginning of their career at sea.

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Passenger Comfort in Commercial Air Travel with Reference to Motion Sickness

Ludwig G. Lederer, Ph.D., M.D., and George J. Kidera, M.D.†*

The greatest discomfort experienced by passengers in commercial air travel is a form of motion sickness commonly referred to as airsickness. The incidence of airsickness depends on such factors as type of plane, weather conditions, etc. Although it is generally very low (approximately 0.5 per cent) it acts nevertheless as an important deterrent to those affected. Another peculiarity seen in commercial air travel is the unusual predisposition to airsickness of the female adult. Various speculations have been made regarding this phenomenon, but few are supported by fact. It certainly is true that there is no essential difference between the sexes in the anatomic make-up of the organs of equilibrium. We feel that it is entirely a matter of susceptibility to the effects of motion. The average adult female is essentially a very sedentary individual. She is not exposed to motion as much as the average male. Her semicircular canal system is relatively undisturbed by such forms of motion as scend, yaw, roll, and pitch. Table I summarizes the incidence of various forms of discomfort among more than one million passengers on commercial airlines. The data were obtained in 1946 and 1947 before the advent of the newer motion sickness remedies. Over three quarters of all cases of discomfort were caused by airsickness. An average of 59 per cent of all persons becoming airsick were women, only 24 per cent men, and the remaining 17 per cent were children.

It is interesting to note the constancy of female airsickness from month to month (54.8 to 62.6 per cent). Supplemental studies made in 1949 show very little change in the distribution of motion sickness among males, females, and children (table II). One important figure in table II is the incidence of motion sickness when it occurs in one airplane. We ordinarily speak of incidence of motion sickness as a system-wide figure; in that case, it is less than 1 per cent of passengers carried. However, when motion sickness strikes in any one airplane the incidence is approximately 8 per cent of those aboard. Table II also shows that the four-motor aircraft DC-4 has a lower incidence of motion sickness than the two-motor smaller aircraft, the DC-3. Most of the motion-sickness flights occurred during daylight hours. This may be a reflection, in part at least, of the visual factor. The lowest incidence was in the evening, when wind currents usually abate. Mixed turbulence is more effective than steady turbulence in causing motion sickness.

Another important factor is the position within the aircraft. It is apparent from table III that the greatest amount of motion sickness occurred consistently in the left-hand window seat of this type of aircraft. Also, seats 14 and 17, situated immediately aft of the trailing edge of the left wing were the most vulnerable. In these seats the

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TABLE 1

Study of Discomforts Encountered in Commercial Aircraft Flights
December 1946 to November 1947

	Dec. (1946)	Jan. (1947)	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	For the Year
A Number of passengers carried	96,476	63,104	58,972	79,019	102,317	119,466	99,032	98,063	112,380	109,928	103,988	78.232	1,120,977
B % Discomfort of all trips flown	27.4	13.41	19.04	26.1	26.01	19.78	16.72	17.50	16.5	12.39	12.98	16.03	18.0
C % Discomfort among passengers carried	1.24	1.10	1.59	2.09	1.68	1.13	1.12	.95	.724	.955	.551	1.25	1.16
D % Airsickness of discomforts reported	68.70	71.87	78.46	81.20	86.61	89.04	83.18	81.11	74.80	69.23	55.40	71.86	77.37
E % Males airsick	26.20	27.80	31.00	31.30	25.99	23.55	15.84	15.52	17.82	23.50	26.55	23.33	23.70
F % Females airsick	59.30	58.10	56.60	55.30	60.13	62.60	62.55	59.65	58.16	59.75	54.82	57.69	59.19
G % Children airsick	14.30	14.00	12.30	13.20	13.87	13.84	21.59	24.82	24.01	16.74	18.62	18.96	17.06
H % Earaches of all discomforts reported	7.34	11.97	5.75	4.10	3.70	3.75	5.63	3.96	7.37	8.85	8.35	7.74	5.99
I % Headaches of all discomforts reported	10.10	11.97	6.18	5.13	4.28	5.22	6.44	11.15	14.00	12.95	19.16	7.23	8.32
J % Temporary deafness of all discomforts reported	8.76	3.71	3.94	3.32	3.41	1.02	1.43	1.28	1.71	3.23	5.92	3.36	3.24
K % Colds and other medical conditions of all discomforts reported	5.59	7.31	5.65	6.22	1.96	.95	3.30	2.46	2.08	5.71	11.11	9.78	5.05

TABLE II

Report of One Month of a Three-Month Trial

CONDITIONS OF EXPERIMENT

Type of Plane	Time of Day	Weather
DC-3 62%	Day — 88%	Turbulent — 29%
	Evening — 3%	Mixed — 55%
DC-4 38%	Night — 9%	Smooth — 16%

SUBJECTS OF EXPERIMENT

Number Airsick Out of 3375 Passengers Carried — 270 Incidence — 8%

Females — 65% (176)	Males — 21% (56)	Children — 14% (38)

passenger is subjected to a quick scanning type of view which induces a nystagmic effect. We believe this initiates a form of vestibular excitation. The left side of the aircraft is more prone to induce motion sickness since banks and turns are usually made in that direction because of the greater visibility afforded the captain, who sits in the left seat of the cockpit.

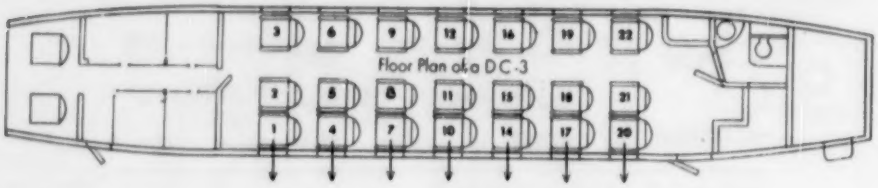
When our studies were initiated in 1946 there was no effective motion sickness prophylactic or remedy available. We used a very ineffective form of "bitters" as a stomachic in those who became sick and had vomited. We began a search of drugs that were then recommended, such as some hyoscine derivatives, some antacids, and even a mint-odor inhaler. In 1949 we tried Dramamine in 100 mg. doses, after Gay and Carliner had reported beneficial results in seasickness. In experiments by Lederer, the incidence of drowsiness was 10 per cent. One of the large airline companies then began using Dramamine in doses of 50 mg. The comparison of tables IV and V shows the relative loss of effectiveness. It was surprising to find only approximately 5 per cent loss of effectiveness with the dose level cut to 50 mg.

Perhaps one of the most interesting studies was done by Kidera in a select group of motion-sickness-prone passengers taking Dramamine prophylactically. This is shown in table VI. It proves that efficacy of treatment is raised from an average of 62 per cent when symptoms of motion sickness are present to about 90 per cent when Dramamine is taken prophylactically.

Two factors have been introduced recently in the field of commercial air transportation which will influence profoundly the comfort of passengers aloft. One of these factors is the actual comfort of the airplane itself. Modern airliners, with temperature control, good soundproofing, adequate ventilation, air foam-cushion seats, cabin pressurization, and high wing-loading surface to reduce effects of motion, are really

TABLE III

Frequency of Airsickness by Seat Numbers—DC-3
From Dec. 1946 through Nov. 1947



Most frequent cases of airsickness with seat locations were observed as follows:								
	Frequency:	258	312	266	298	333	343	221
	Percentage:	6.0%	7.2%	6.2%	6.9%	7.7%	8.0%	5.0%

Motion sickness was distinctly greater in incidence in the left-hand window seat of the DC-3 type airplane, as shown in this diagrammatic sketch of the seating arrangement of the DC-3 with percentage of airsickness seen in each seat indicated. The data for these percentages are based on well over 5,000 cases of motion sickness.

Less Frequent Cases of Airsickness

Seat Number	Frequency	Percentage
18	184	4.3%
15	182	4.2%
12	181	4.2%
19	177	4.1%
16	171	4.0%
6	170	3.9%
2	167	3.9%
5	167	3.9%
11	154	3.7%
3	152	3.5%
21	146	3.4%
9	141	3.2%
22	128	2.9%
8	123	2.8%
	4,274	

conducive to comfortable travel and obliterate many physical effects that used to predispose people to developing motion sickness. Kidera reports that in the new DC-7 aircraft on coast-to-coast flights, where navigation aids have reduced effects of turbulent weather to a minimum, there has been no case of airsickness reported in these 60 to 80 seat airliners in over six weeks of operation.

We will always have with us, however, the susceptible passenger and smaller aircraft. To these, the second great contributing factor to comfort is the introduction of the benadryl-type antihistamine and some of the cyclizine compounds. Dramamine, with its diphenhydramine base, and other remedies such as Marezine, Bonamine,

TABLE IV

Effect of 50 mg. of Dramamine

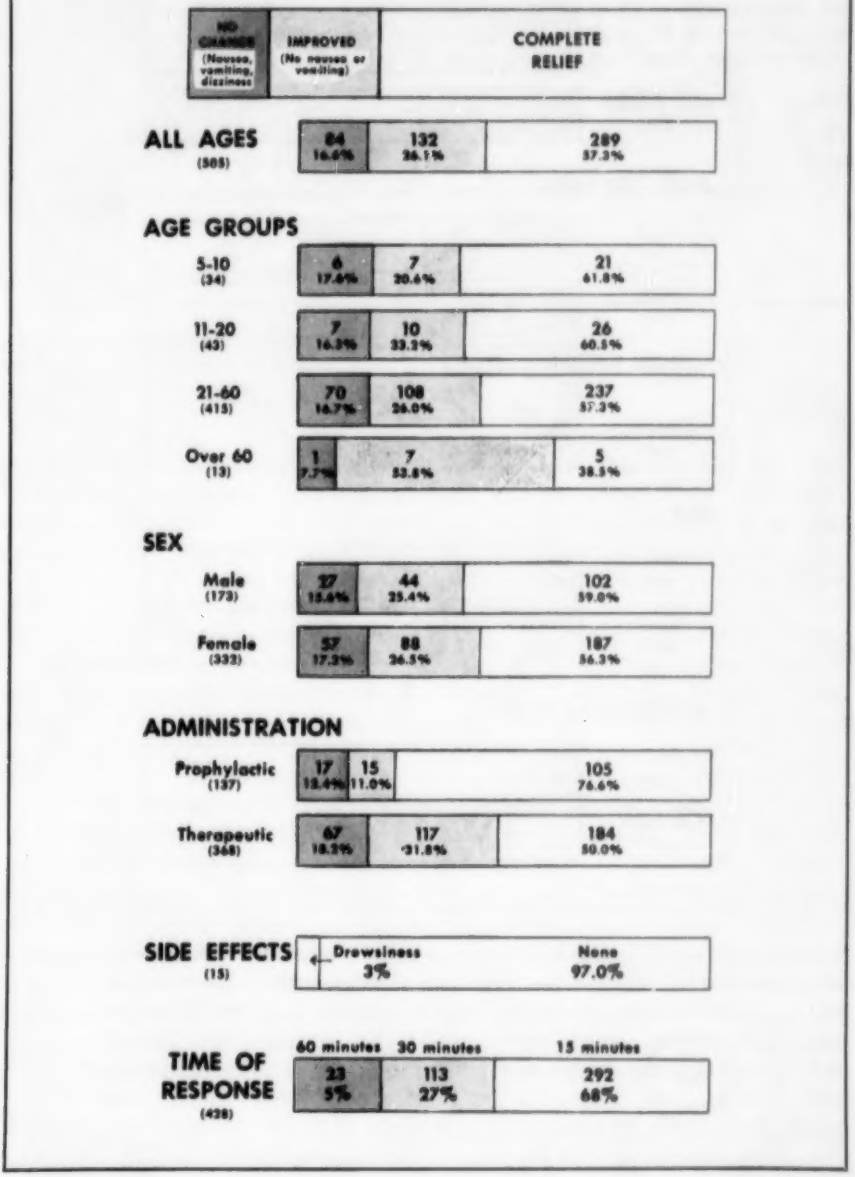


TABLE V

Effect of 100 mg. of Dramamine

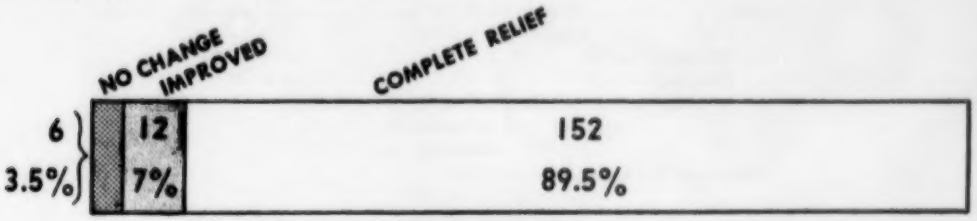
	NO CHANGE (Nausea, vomiting, dizziness)	IMPROVED (No nausea or vomiting)	COMPLETE RELIEF
ALL AGES (2129)	286 14%	518 24%	1322 62%
AGE GROUPS			
5-10 (157)	22 14%	34 22%	101 64%
11-20 (155)	27 17%	34 22%	94 61%
21-60 (1711)	222 13%	416 24%	1071 63%
Over 60 (106)	15 14%	34 32%	57 54%
SEX			
Male (554)	76 14%	128 23%	350 63%
Female (1575)	210 13%	390 25%	975 62%
ADMINISTRATION			
Prophylactic (536)	24 5%	60 11%	449 84%
Therapeutic (1593)	262 16%	458 29%	870 55%

Benacine, etc., are all enjoying widespread use as a form of treatment and preventive for motion sickness. Lederer has tried many different drugs of various types. We have used scopolamine alone, Scopodex, Trip-eze (another scopolamine derivative), procaine amide, Mosidal, etc. In general, we feel that the scopolamine derivatives alone, without antihistamine combination, is not too effective once the symptoms of motion sickness are elicited. They are rather effective prophylactically, but in certain susceptible individuals the scopolamine side effects of mental aberration are not desirable. They certainly are not the drug of choice for medicating children. Recently,

TABLE VI

Effect of Dramamine Prophylaxis

During the month 170 passengers volunteered the information that they were unusually susceptible to motion sickness or had been sick on a previous flight. To these passengers, one dose of Dramamine was given as a preventative while still on the ground prior to boarding the plane, at the time of takeoff, or during flight while the plane was approaching possible turbulent air. The results of this breakdown are as follows:



Tuttle* states in conclusion: "The six cases by whom no relief was experienced evidently could not retain the Dramamine in their stomachs long enough to get it into the blood stream because of incessant vomiting. . . . The prophylactic value was conclusively demonstrated."

559 cases of airsickness were treated after the symptoms had become manifest. The following drugs were each used in approximately the same number of cases: Bonamine, Benacine, Dramamine, Marezine, Trimeton, and Seda-Stay (sodium bromide, scopolamine hydrochloride, and thiamine hydrochloride). All drugs seemed rather effective, although in this small series Trimeton (25 mg.) was best and Dramamine the poorest. The former compound relieved 91.1 per cent of the cases and the latter 72.3 per cent. The remaining preparations ranged between these extremes with an over-all relief of 92.1 per cent. In 255 subjects the drugs were used prophylactically, and 93.9 per cent failed to become airsick. Benacine (25 mg.) gave the poorest protection (80.1 per cent), while Marezine (50 mg.) protected all subjects receiving it (0.0 per cent sick). Marezine seems especially promising, since it is markedly effective and has little or no side effects such as drowsiness. Another advance in treatment is the introduction of rectal suppositories of Dramamine and Marezine, which are a boon to the passenger who has developed repetitious vomiting and cannot retain oral medicine.

Steps are now being taken by most of the manufacturers to have motion sickness remedies released to the public by over-the-counter sale. We feel this is advisable because it will permit premedication, which is the most effective way to combat motion sickness. Table VII shows a study of the effectiveness of Marezine (cyclizine HCl) in a small selected group of airsickness in DC-3 type aircraft flown over routes well known for their turbulent air lanes.

Air travel in recent years has opened new vistas of travel to *all* segments of the population. With the advent of more equipment, larger aircraft, and coach type of air

* Lederer, L. G., Kidera, G. S., and Tuttle, A. D.: Medical aspects of passenger flight in commercial airlines. Presented at the Aero Medical Association Convention, New York, N. Y. (Aug. 31) 1949.

TABLE VII

Marezine

50 mg. Dose Orally on DC-3 Type Aircraft Flying Routes Known to Cause Airsickness

Number of Flights Showing Motion Sickness		29	100%
	Daylight Flights—	26 =	89.9%
	Night Flights—	3 =	9.1%
Weather:	Smooth— 5 = 17.2%		
	Mixed— 16 = 55.0%		
	Rough— 8 = 27.6%		
Total Passengers Carried		381	100%
Passengers Airsick		93	22.4%
Motion Sick Passengers:	Males—	22 =	23.6%
	Females—	61 =	65.6%
	Children—	10 =	10.7%
Symptoms in Those Already Motion Sick:			
	Sweating—	25 =	100%
	Relieved—	21 =	84%
	Nausea—	31 =	100%
	Relieved—	24 =	77.5%
	Vomiting—	14 =	100%
	Relieved—	13 =	93%

travel, this form of conveyance is no longer restricted to the financially elite. This means that many unselected patients with illness or organic disease are eligible for air passage. Some illnesses, such as organic heart disease in cases of healed infarcts, may be aggravated by episodes of vomiting such as occur in severe airsickness. The proper and effective treatment of airsickness thus becomes not only a necessity for comfort but also a prophylactic measure against the aggravation of pre-existing organic conditions.

SUMMARY

A review has been presented of the incidence of a form of motion sickness peculiar to air travel. Data have been accumulated to show that airsickness, although declining, is still the greatest source of passenger discomfort. Interesting facts, such as seating arrangements in aircrafts, which cause more airsickness, and the unusually high susceptibility of the adult female to airsickness, have been demonstrated. A review of the several categories of effective motion sickness remedies has been presented. The premedication effect has been stressed, showing that prophylactic treatment is much more effective than treatment after symptoms are manifest. The relationship of airsickness aggravating pre-existing organic conditions such as heart disease has been mentioned to show a need for proper treatment of one to avoid aggravating the other in unselected air passengers.

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INCORPORATING INTERNATIONAL RECORD OF OPHTHALMOLOGY

FOREWORD

The objective of the QUARTERLY REVIEW OF OPHTHALMOLOGY is to bring together in one publication concise but authoritative abstracts of current articles on ophthalmology, according to an all inclusive plan which includes all special, state, and national journals as well as the bulletins of clinics, hospitals, etc., and the transactions of meetings. This embraces both the domestic and foreign literature.

To assist the reader to locate quickly the articles of current interest all data are classified and published according to the following systematic plan:

Anatomy, Embryology, Heredity, Development and Nutrition

Optics, Physiology and Psychology of Vision

Physiology, Chemistry and Biochemistry of the Eye

Pathology, Bacteriology and Immunology

Diagnostic Methods of Examination Biomicroscopy and Photography

Ocular Movements and Motor Anomalies, Nystagmus, Reading Disability

Anomalies of Refraction and Accommodation, Contact Lenses

Conjunctiva

Cornea, Sclera and Tenon's Capsule

Anterior Chamber and Pupil

Uveal Tract and Sympathetic Ophthalmitis

Crystalline Lens

Vitreous Humor

Retina

Neuro-Ophthalmology, Optic Nerve, Visual Pathways, Centers and Visual Fields

Eyeballs, Exophthalmos and Enophthalmos

Glaucoma and Hypotony

Lacrimal Apparatus

Eyelids

Orbit

Allergy

Anesthesia

Medical Ophthalmology

Pharmacology, Toxicology and Therapeutics

Comparative Ophthalmology

Tropical Ophthalmology

Hygiene, Prophylaxis, Occupational Ophthalmology and Injuries

Illumination and Illuminating Engineering

Ophthalmic Sociology

Education, History and Institutions

Miscellaneous

Book Reviews

Announcements

A section entitled International Record of Ophthalmology is included at the beginning of the journal. The Record Section consists of advanced clinical and experimental reports.

Quarterly Review of **OPHTHALMOLOGY**

ABSTRACTS

OPTICS, PHYSIOLOGY, AND PSYCHOLOGY OF VISION

73. *Effect of Various Durations of Red Adaptation on the Course of Subsequent Dark Adaptation.* MILTON S. KATZ, AILENE MORRIS, AND FORREST L. DIMMICK. Medical Research Laboratory, U. S. Naval Submarine Base, New London, Conn. Report No. 246, April 27, 1954.

The experimental data in the report pertains to the optimal duration of red light, the conditions under which it may be preferable to darkness, and the ensuing changes in dark adaptation. The data also represents substantiation of earlier reported results.

Thresholds were determined with 2 degree peripheral stimuli of two-tenths of a second duration through the course of dark adaptation. Experimental photopic-adaptation brightnesses were 16 foot lamberts of red light for 10 minutes.

Experiments yielded the following results: (1) Red adaptation results in a more rapid rate of early subsequent dark adaptation than does white light of the same brightness. (2) After 15 minutes of dark adaptation, a relatively stable level of sensitivity is reached which is only slightly affected by preadaptation conditions. After 30 minutes, the thresholds give no evidence of differential effect. (3) No duration of red adaptation results in as low a threshold as a like duration of darkness.

PHYSIOLOGY, CHEMISTRY, AND BIOCHEMISTRY OF THE EYE

74. *The Filling-In Process.* GORDON L. WALLS. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:329–341, July 1954.

The physiologic blind spot, the normal scotopic central scotoma, König's foveo-central blue-blind spot, and the early scotoma of the migraine aura are all filled in subjectively with whatever visual material surrounds them in the field. They are all "negative" exactly like scotomata from lesions of the primary visual cortex. It does not seem biologically possible that an individual could be unaware of such scotomata. A general physiologic filling-in process, however, appears to be demanded by the properties of optic nerve fibers. These conduct importantly only at "on" and "off." The perimeter of a homogeneous target is continuously registered by on and off bursts owing to the physiologic nystagmus. It is proposed that a special cerebral process fills in the area within such a contour with whatever

activity is at the contour. The fact that this process necessarily operates upon many scotomata to make them negative is "purely coincidental."—*Author's abstract.*

PATHOLOGY, BACTERIOLOGY, AND IMMUNOLOGY

75. *Experimental Intra-ocular Infection with Mumps Virus.* H. P. KIRBER AND MARIA WIENER KIRBER, Philadelphia, Pa. Arch. Ophth. *51*:832–849, June 1954.

The Enders strain of mumps virus produces a keratitis with iridocyclitis in guinea pigs on inoculation into the anterior chamber. The typical clinical and histologic lesions and variations in the course of the infection are described. A brief review of the published cases of clinical mumps keratitis is presented and the relationship of the experimental infection to the clinical course in man is discussed.

Experimentally, keratitis and iritis are produced only by the injection of active, living virus into the anterior chamber, not by virus inactivated by heat or ultraviolet irradiation. Guinea pigs injected into the anterior chamber with active mumps virus produce high antibody titers against virus and soluble antigens of mumps. Antibody titers elicited by inoculation of inactivated mumps virus, or of active mumps virus by conjunctival deposit, corneal scarification, intradermal or footpad inoculation, are consistently lower. Guinea pigs tested with mumps skin test material at 1 month, 11 weeks after the original contact with mumps virus, gave a negative skin test. Guinea pigs with low or no antibody titer after the original contact with the inactivated mumps virus or with active mumps virus by any route responded to the skin test with high antibody titers.

The relationship of these findings to similar observations in man is discussed. The Enders strain of mumps virus produced no lesions in rabbits on inoculation into the anterior chamber of the eye. No demonstrable complement-fixing antibodies against mumps antigens were produced in these animals. 25 references. 14 figures.—*Author's abstract*

76. *Ocular Complications Associated with Malaria.* J. V. M. ROSS. Eye, Ear, Nose and Throat Monthly. *32*:707, December 1953.

Malaria and its treatment are associated, at least chronologically, with many ocular disturbances. A possible common pathologic basis, vascular obstruction, is discussed for all ocular complications due to the disease process. Eye lesions, predominantly of the cornea, developing during the acute phase of malaria are usually monocular, moderately incapacitating, and may leave permanent sequelae. Those occurring in the chronic phases are more likely to be bilateral, involving the posterior segment. They are apt to result in final and more serious visual damage. Treatment with antimalarial drugs has been generally successful. However, herpetic ocular disturbances, severe intra-ocular and ocular neurologic

lesions, and the complications of the therapy itself, do not respond to antimalarial treatment.

DIAGNOSTIC METHODS OF EXAMINATION, BIOMICROSCOPY AND PHOTOGRAPHY

77. *Use of Radioactive Phosphorus in Detection of Intra-ocular Neoplasms.* I. J. EISENBERG, I. H. LEOPOLD, AND D. SKLAROFF, Philadelphia, Pa. Arch. Ophth. *51*:633–641, May 1954.

A method is described whereby intra-ocular neoplasms are detected by taking a count of the concentration of radioactive phosphorus (P^{32}) in both eyes, one hour after the intravenous injection.

The per cent variation of the highest reading in the normal eye, from the average of the normal eye, may run as high as 28 per cent though generally less than 20 per cent. A variation of 30 per cent or more between the average reading of the normal eye, and the highest reading of the pathologic eye, should be considered a significant concentration of P^{32}. Serious detachments, and choroidal and vitreous hemorrhages without signs of inflammation, show no significant concentration of P^{32}. Inflammatory lesions, if active, will show a significant concentration of P^{32}.

Neoplasms in the posterior segment of the globe may or may not show a significant concentration of P^{32}, depending on their size, activity, and location. Neoplasms in the anterior segment of the globe show a significant detectable concentration of P^{32}. This test will prove of value when used in addition to other diagnostic methods for detecting intra-ocular neoplasms. 8 references. 6 figures. 2 tables. —*Author's abstract.*

OCULAR MOVEMENTS AND MOTOR ANOMALIES NYSTAGMUS READING DISABILITY

78. *The Role of Occlusion in Amblyopia Correction.* ABRAHAM SCHLOSSMAN, New York, N. Y. Eye, Ear, Nose & Throat Monthly. *33*:305, May 1954.

Occlusion is the most effective treatment for amblyopia and is well within the scope of any ophthalmologist even if he has no orthoptic instruments available. The most favorable cases are those in which the loss of central vision is apparently secondary to a convergent strabismus. The time to begin occlusion for the improvement of central acuity is at the first indication of poor vision.

Total occlusion with an elastoplast bandage has given the best results. Parents should be cautioned that the angle of squint may increase while a child is wearing the patch, because the latent element of the strabismus becomes manifest. Occasionally, visual acuity in the fixing eye may decrease, but it usually improves quickly after occlusion is discontinued.

While the patient is wearing the occluder, it is advisable for him to perform certain tasks to help improve his vision. In fact, any method which forces the

patient to use his amblyopic eye while the other one is occluded is helpful in improving vision. After the visual acuity of the amblyopic eye has become equal to that of the other eye, the patient should be trained in fixation with the usually deviating eye even after the patch is removed. Often a patient who has learned to hold fixation with the deviated eye will complain of diplopia.

In uniocular convergent strabismus, amblyopic training should be begun before any surgery is contemplated. If the vision does not improve after two months, the necessary surgery should then be performed. If the amblyopic exercises are successful, surgery may be postponed until the vision is equal in both eyes, or until maximal improvement is obtained. After surgery, partial occlusion or other orthoptic training may be resumed if necessary.—*Author's abstract.*

79. *A Case of Ocular Torticollis.* J. AUDET. Canad. M. A. J. *71*:60–62, July 1954.

Torticollis may be congenital, spasmodic, secondary, or ocular. When the cause is ocular, a vertical squint is present when the head is straightened. A torticollis in a 4 year old boy was immediately and completely cured by tenotomy of the right inferior oblique. 3 references. 3 figures.

80. *The Cross-Eyed Child.* R. D. HARLEY. M. Times, New York. *82*:85, February 1954.

Although many people, including some physicians, are of the opinion that nothing can be done for strabismus until the child is old enough for subjective examination, the ideal time to begin correction is immediately after recognition of the condition. At about the age of 6 months the eyes should begin to show coordination and parallelism. If the child's eyes remain crossed beyond this time attention is necessary. If left unattended, the eyes will show some permanent defect.

Correction of the resulting amblyopia may involve occlusion of the good eye to force the poor eye to work, prescription lenses, eye exercises, surgery, or a combination of these measures. When surgery is indicated, it is essential that it be accomplished as soon as possible. Best functional results are achieved before the child has entered school. If muscle surgery is delayed the eyes can still be straightened satisfactorily but the functional result is not good.

81. *The Dependence of Cyclophoria on Convergence Elevation and the System of Axes.* MERRILL J. ALLEN. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:297–307, June 1954.

Cyclophoria is generally the deviation of the vertical meridians of the retinae away from parallelism and may normally be as high as 16 degrees. The classic data of Landolt, Hering, Donders, and others are presented and discussed. These data are compared with those that might be expected if the cyclophoria could be explained by Listing's law of monocular eye movements.

The problem of "false torsion" and its relationship to the system of axes of

Helmholtz, Fick, and Listing are considered. The differences in the system of axes of such instruments as the synoptoscope and the orthorater account for the wide differences in cyclophoria obtained with these instruments. A formula and graph for converting synoptoscope data (Fick's system of axes) to the Helmholtz system of axes is presented to illustrate the interchangeability of data from one system of axes to another.

Some speculation is given as to the role of the third nerve nucleus and the inferior obliques in the production of cyclophoria associated with convergence. A small amount of original data is presented along with consideration of the practical implications of cyclophoria in eye testing and instrument design. 12 references. 10 figures.—*Author's abstract.*

82. *Occurrence of Slight Facial Malformations in Cases of Strabismus.* DR. A. URRETS-ZAVALIA, JR. Presented at the International Congress of Ophthalmology, New York. September 1954.

Convinced that imbalance of the muscles that control the upward movements of the eyes, thus causing squint, is the result of imperfect prenatal development, the author studied persons with this type of squint in an attempt to determine whether they show other anomalies of growth.

As a result of his observations, he found that some minor defects of the bones around the eye and the adjacent cheek bones were consistently associated with the presence of irregularities in the muscles. Although he has not as yet attempted to investigate them from the point of view of genetics, findings have indicated that the defects of bone and skin were common among otherwise normal relatives of the patients with strabismus.

It is not clear whether these anomalies are due to a mutated gene and are of a transmissible nature, whether they depend on a defect of the cytoplasm of the egg, or on a fortuitous, extrinsic, peristatic influence acting upon the embryo at an early developmental stage.

One peculiarity frequently seen was a slanting of the eyelids, in a direction opposite to that found in the Mongoloids. This has been described as an "anti-Mongoloid obliquity." With this anomaly, the lower lid is sometimes found to have a shape similar to the letter "S."

ANOMALIES OF REFRACTION AND ACCOMMODATIONS, CONTACT LENSES

83. *A Second Report on the Fitting and Use of Microlenses.* JOHN COLLINS NEILL. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:411–415, August 1954.

The author describes his work with ultrathin corneal contact lenses, which he calls "microlenses." The original use of plastic lenses as thin as 0.1 mm. (0.004 in.) proved unsatisfactory because of a lack of rigidity in the plastic material.

However, with the plastics now available, the optimum thickness of a microlens lies between 0.18 mm. and 0.20 mm. The most satisfactory diameter for 90 per cent of patients is 9.5 mm. In practice, the microlens must be fitted even flatter than the Tuohy corneal lens to permit proper "lag" during eye movements. In 75 per cent of cases, the fitted lens was 3.5 diopters flatter than the flattest corneal meridian.

In this paper, the author recommends the use of the keratometer for measuring corneal curvatures, although satisfactory fittings can be made by observation of "lag" and the use of fluorescein. Forty per cent of the cases fitted successfully with microlenses had previously been fitted unsuccessfully with other types of contact lenses. The office procedures for fitting microlenses are also described.—*Author's abstract.*

84. *Stereophotographic and Stereophotogrametric Reproduction of the Cornea and Sclera of the Living Eye.* J. RZYMKOWSKY. (TRANS. BY WILLIAM P. SCHUMANN). Am. J. Optom. & Arch. Am. Acad. Optom. *31*:416–422, August 1954.

The fitting of a contact lens by stereophotogrametric means is presented. The contact lens is of the scleral rim variety, and while at present not as much used as the corneal lens, its use might still be indicated in certain cases. Further studies along these lines should also be continued.

The apparatus employed was a simple stereoscopic camera 6 cm. by 9 cm. and a separation of 6.5 cm. To obtain a good picture of the living eye flashbulbs were used, which did not cause any discomfort to the patient. The negative was evaluated by means of a stereoscope, and from the result, the contact lens manufactured. For greater accuracy, an instrument, such as is used for aeronautical mapping, is advisable for stereoscopic interpretation and measurement.

All other materials are manufactured in the United States, eliminating a dependency on foreign material.—*Author's abstract.*

85. *The Ciliary Body in Accommodation and Accommodative-Convergence.* MEREDITH W. MORGAN, JR. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:219–229, May 1954.

The detailed haploscopic studies discussed in this paper indicate that the ciliary body acts like an elastic body and that over most of the range of accommodation no more effort is required for each succeeding diopter than the preceding one. There is also evidence that the subject measured has a considerable physiologic reserve of accommodation tending to support the theory of Flieringa and van der Hoeve. It has been determined that convergence acts as a stimulus to accommodation and that for one subject the values of the accommodative-convergence accommodation ratio, and the convergence accommodation ratio are different.

86. *Effect of Cigarette Smoking on Flicker Fusion Threshold; Clinical Observations on Normal Smokers and Nonsmokers.* LAWRENCE L. GARNER, E. FRANKLIN CARL, AND ERWIN E. GROSSMAN, Milwaukee, Wis. Arch. Ophth. *51*:642–655, May 1954.

Using a Krasno-Ivy flicker photometer, observations were made of the effect of smoking on the flicker fusion frequency of 93 healthy student nurses. Twelve additional older subjects were also employed, ranging in age from 24 to 53 years.

Binocular foveal fixation was employed at a distance of 1.5 meters under standard conditions of illumination and exposure. For 12 hours prior to any flicker determinations, the subjects were required to abstain from smoking. Repeat tests were performed on all subjects. Of the 108 persons tested, 35 revealed an increase in the flicker fusion frequency after smoking; 21 revealed a decrease; 40 presented no alteration in the flicker fusion frequency after smoking; 5 presented an initial rise followed by a secondary fall in the flicker fusion rate, and 7 subjects gave erratic results. The use of a denicotinized cigarette would eliminate the rise of flicker fusion frequency in the initial rise group and would either reduce or eliminate the drop in the group presenting the marked drop in flicker fusion frequency following smoking an ordinary cigarette. Permitting the subject to sit in a smoke filled room resulted in a drop similar to that produced by smoking. This finding made it necessary to conduct all tests in well ventilated rooms while permitting all smoking to be done in adjacent rooms. Glyceryl trinatrate, 1/100 grain (0.6 mg.), taken sublingually produced a prompt return to the flicker fusion threshold base line, indicating its mode of action through its vasodilatory properties. Normal recovery time for the return to the presmoking base line varied from 4 minutes to as long as 50 minutes. The longest recovery times were noted in the "drop" group.

An interesting observation was the effect of noise on the efficiency of the tests. In each instance, a decided drop in flicker fusion frequency was noted, the rate becoming erratic for one to three minutes, then returning to a level near the last prenoise reading. Fundus studies using the ordinary Morton head ophthalmoscope failed to reveal any evidence of retinal vascular changes during these tests.

Twelve of the subjects employed showed a variability of findings, which in 7 subjects made it impossible to consider them as candidates. It is possible that these variations might be eliminated by the use of a stroboscopic type of apparatus which is considered more accurate by Dr. Miles. Findings indicated that in some subjects there exists a hyperreactibility to nicotine. This hyperreaction varies from mild to extreme and can in no way be predicted, nor is it related in any way to past smoking habits. It is assumed that the drop occurring, following the act of smoking, is based on vasoconstriction anoxia since it is easily reversible by the use of dilators. However, this does not explain the acute rise noted in the initial rise group, unless one postulates an initial stimulating effect later masked by its vascular effect. The flicker fusion threshold response offers a potentially practical method of detecting hyperreactions to nicotine and should become of value to the clinician. 16 references. 6 figures. 4 tables.—*Author's abstract.*

87. *Changes in Refraction during Life.* M. BUCKLERS. Brit. J. Ophth. *37*:587, October 1953.

In hyperopic persons the curve of refractive error shows a steeper ascent during the first two decades than after the period of growth. After about 20 years of age, the changes in refraction are usually small.

In general, a quick and steady increase of myopia may be expected when the onset is early and of a high degree. However, exceptions have been noted. In some cases, myopia becomes constant early; this may apply to low as well as high myopia. Sudden and distinct increases of myopia have been noted occasionally in cases that had remained stationary up to the fifth and sixth decades of life. Instances of lessening of myopia during the course of a lifetime have also been noted. Hyperopia is generally stationary in its course, although steeper increases have been noted for a few years in youth than anytime in later life. It has been demonstrated that the change in refraction for each eye is determined throughout life by endogenous factors and can not be explained by external influences.

CORNEA, SCLERA, AND TENON'S CAPSULE

88. *Ballistic Measurements in Corneal Disease Using the Wiegersma Elastometer* DR. A. E. MAMELOK AND ADOLPH POSNER. Presented at the International Congress of Ophthalmology, New York. September 1954.

Doctors Mamelok and Posner state that an instrument known as an elastometer will register differences in the corneas of two eyes. Following up this indication, they report they consistently found such differences pointed to disease.

The elastometer, so called because it measures the elasticity of tissue, was described as follows: "The Wiegersma elastometer is essentially a hammer which consists of an ivory ball two millimeters in diameter mounted on the end of an arm 150 millimeters long. The hammer is mounted in a jeweled pivot in the center of a calibrated quadrant and is counterbalanced so as to be extremely light. The resting position of the hammer is in the horizontal plane. When the hammer is raised to the vertical position and then released by a tripping mechanism, it traverses an arc of 180 degrees and then returns to its original position, less a small amount lost by friction. If an object is struck by the ball the instant it reaches the horizontal position, the hammer rebounds upward. The amount of rebound is measured on the quadrant of the elastometer."

Although the instrument devised by Wiegersma was the most efficient of several intended to measure pressure within the eye by means of the ballistic principle, it still was not consistently successful. As a result of their experience with it, however, they believe it may be useful for the study of diseased corneas, and especially for measuring the healing and integrity of transplanted corneas.

UVEAL TRACT AND SYMPATHETIC OPHTHALMITIS

89. *Capillary Tube Method for Antibody Determination; Preliminary Studies in Aqueous of Rabbits Sensitized with Various Antigens.* TED SUIE AND JOSEPH SUDIMACK, JR., Columbus, O. Arch. Ophth. *51*:875–877, June 1954.

The small amount of aqueous humor which is available for titration of antibodies is probably the chief factor which precludes the study of the ocular immunologic manifestations in sensitized animals. A capillary tube method, therefore, has been applied whereby small quantities of an aqueous humor sample may be titrated against a series of antigens. The presence of antibodies in the aqueous of animals sensitized with bovine albumin and micrococcal vaccine, respectively, were demonstrated by this procedure and by conferring dermal sensitivity to normal rabbits with secondary aqueous humor. The practical application of such a diagnostic method in cases of nongranulomatous uveitis is presented. 2 references. 1 table.—*Author's abstract.*

90. *Antihyaluronidase and Antistreptolysin Titers in Uveitis.* IRVING H. LEOPOLD AND THOMAS G. DICKINSON, Philadelphia, Pa. Tr. Amer. Acad. Ophth. *58*:201–211, March-April 1954.

The study of the etiology of uveitis has long presented one of the most vexing problems with which the ophthalmologist has had to contend. Inflammatory conditions of the uveal tract can be generally classified into three etiologic groups: those caused by injury, those caused by direct extension of inflammatory conditions of other parts of the eye, and those which are considered to be the result of endogenous infections and their sequelae.

Woods has further classified this third group into two main subdivisions—granulomatous and nongranulomatous. Of the granulomatous uveitis, the major causes are thought to be syphilis, tuberculosis, sarcoid, brucellosis, toxoplasmosis, various fungus infections, nematode infestations, and virus diseases. In these, there has been occasional evidence of actual invasion of the uvea by the causative organism. In the nongranulomatous uveitis, the cause is thought to be a sterile allergic reaction or a hypersensitivity of the ocular tissues to a specific bacterium or its products. The hemolytic streptococcus and the gonococcus have been suggested as the principal offenders.

It is characteristic of the growing hemolytic streptococcus to liberate into the surrounding tissue a number of independently acting extracellular substances. The four most important of these are: streptococcal fibrinolysin or streptokinase, streptococcal desoxyribose nuclease or streptodornase, streptococcal hemolysin or streptolysin, and streptococcal hyaluronidase.

These four extracellular substances liberated by the hemolytic streptococcus plus two fractional parts of the streptococcal cell; namely, the cytoplasmic particles and the supernatant or nuclear fraction, being protein in nature, can act as antigens and produce specific antibodies in the blood stream. These antibodies, produced

as a consequence of widely prevalent human contact or infection with the streptococcus, have a characteristic age distribution. The antihyaluronidase and antistreptolysin titers were measured in a random series of cases of uveitis in order to learn if any apparent relationship could be demonstrated between this disease and streptococcal exotoxins in the body. Sixty-three cases were chosen from the ward services of the Wills Eye Hospital.

Statistical analyses applied to the results suggest that the cases of uveitis in this study do not represent a normal group with regard to serum antihyaluronidase and antistreptolysin titers. These data also suggest that the hemolytic streptococcus may play an etiologic role in the production of uveitis. The data go on to indicate that these tests, particularly the antihyaluronidase titers, should be studied further for a possible method of detecting the etiology in cases of uveitis. 23 references. 9 tables.—*Author's abstract.*

CRYSTALLINE LENS

91. *Cataract and Its Etiology.* ARTHUR SHLAIFER. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:437–458, September 1954.

This paper is a summary of the literature of relatively recent years pertaining to the etiology of cataract. The general pathology and classification of cataract, the metabolism of the lens and its chemistry, and the nonsurgical treatment of cataract are briefly discussed. The relationship between cataract and the following subjects is presented: heredity, refractive errors, lipids, cysteine, galactosemia, calcium, diet, diabetes, radiant energy, electricity, microwaves, roentgen irradiation, and neutrons.—*Author's abstract.*

RETINA

92. *Oxygenation and Retrolental Fibroplasia.* T. H. INGALLS AND N. PURSHOTTAM. New England J. Med. *250*:261, April 15, 1954.

The literature on the etiology and development of retrolental fibroplasia is reviewed. The authors term the condition the end stage of retinal vascularization brought on most frequently in premature infants by excessive oxygen supply in neonatal life.

The onset and severity of the condition are probably influenced by other factors: excessive warmth and humidity, electrolyte shifts, and antioxidants and enzyme derangements, which limit survival and may determine ocular disease in the immature fetus. The mechanism postulated for the development of retrolental fibroplasia is histotoxic anoxia incited by derangement of tissue enzymes at critical periods of fetal development.

Evidence is presented which indicates that the anomaly of the retinal vasculature

has been prevented by careful attention to the optimal supply of oxygen. The authors predict that as the incubator-induced form of retrolental fibroplasia is brought under control, a sporadic form will emerge which depends on a series of circumstances resulting in repeated or prolonged bouts of anoxia and on some of the other factors mentioned.

EYEBALLS, EXOPHTHALMUS, AND ENOPHTHALMOS

93. *Experimental Exophthalmos Produced by Cortisone in Rats; Further Observations.* K. ATERMAN, Birmingham, England and S. M. GREENBERG, Philadelphia, Pa. Arch. Ophth. *51*:822–831, June 1954.

The previous observation of the authors that the prolonged administration of cortisone acetate to young rats produces exophthalmos has been followed up in this paper with particular regard to three questions: (1) Was this phenomenon caused by a hormonally conditioned enlargement of the eyeballs or was it "classic" exophthalmos, i.e., the undue protrusion of a normal eye? (2) Could cortisone produce its effect in the absence of the anterior pituitary gland? (3) Was it possible to counteract this effect by the simultaneous induction of a hyperthyroid state?

To answer the first question the absolute and relative (eyeball weight per 100 Gr. body weight) eyeball weights of intact rats fed various diets and treated with cortisone were determined. Treatment with this steroid regularly produced a significant increase in the relative eyeball weight which, however, could not be correlated with the degree of exophthalmos. In fact, the highest ratios were found in hypophysectomized rats given cortisone, in which exophthalmos was absent. Ocular enlargement or disproportion could therefore not be invoked as the explanation of cortisone-induced proptosis. The failure of cortisone to produce exophthalmos in hypophysectomized rats suggests that another factor, presumably the thyroid-stimulating hormone of the adenohypophysis, must be present in the body if this cortisone effect is to take place. Finally, a comparison of the appearance and of the eyeball weights of rats treated with cortisone and fed increasing amounts of thyroid powder showed a disappearance or a decrease of ocular protrusion, although the eyeball weights showed only minor changes. These findings are considered to support the view advanced by the authors that cortisone-induced exophthalmos is the result of the synergistic effect of the thyroid-stimulating hormone and of an excess of cortisone, and that the thyroid hormone counteracts this effect by raising the animals' metabolic rate. This result preventing the building up of the excess of cortisone necessary for ocular protrusion to occur. 15 references. 2 figures. 3 tables.—*Author's abstract.*

94. *Treatment of Intra-ocular Tumors with Radon.* ARTHUR JOYCE AND R. KAYE SCOTT. Presented at the International Congress of Ophthalmology, New York. September 1954.

A method of treating tumors within the eye with gold radon "seeds" in such a

manner that the radium emanations were evenly distributed is described by Doctors Joyce and Scott.

Because the spaces within the eye are so small, special methods of determining dosage had to be devised. Measurements of the areas to be treated are made with extreme care, and it is the precise placement of the radon seeds, three to six for each tumor, that produces the uniform radiation. The seeds are left permanently in the eye, and the tumors slowly recede.

The authors report they have treated 6 patients in the past 11 years. In 5 patients, the tumors were controlled; 3 of them have now been well for five years. One patient died of heart failure, and one is a recent case. In the sixth patient, the eye was enucleated, but the woman was said to be alive and well. The first patient, a young boy treated in 1943, suffered an injury to his treated eye in 1952, but has recovered.

Summarizing their experience, Doctors Joyce and Scott listed the following advantages of their method: only one operation is needed, several tumors can be treated at once, radon seeds are so distributed that the radiation is uniform, complications have been few and mild, and there has been no serious damage to other parts of the eyes.

GLAUCOMA AND HYPOTONY

95. *Iritis and Glaucoma Associated with Mumps.* RALPH S. RIFFENBURGH, Pasadena, Calif. Arch. Ophth. *51*:702, May 1954.

A case is reported of a 26 year old male who developed a unilateral iritis and secondary glaucoma five days after the onset of mumps, and accompanying an orchitis.

In spite of the use of pilocarpine for two days, the iritis subsided completely with residual change within a week. The improvement of the condition through the use of pilocarpine, as rapidly as that of previously reported cases treated with atropine, suggests the iritis to be self-limited, regardless of treatment.—*Author's abstract.*

EYELIDS

96. *Staphylococcic Eczema of the Eyelids.* F. H. THEODORE. Presented at the International Congress of Ophthalmology, New York. September 15, 1954.

Chronic eczema of the eyelids sometimes results from staphylococcic infection in some part of the eye, according to Dr. Theodore. In recent years, so much attention has been directed to contact allergy as a cause of eczema of the eyelids that the possibility of a bacterial origin is sometimes overlooked, and treatment is thereby made more difficult. While it is true that allergies to drugs and cosmetics are the most common causes of the acute form of eczema on the eyelids, these

substances are less important in the chronic form. Differentiation is important, however, since the treatment given for the acute form is likely to aggravate the bacterial form. When it can be proved that the eczema is related to a condition within the eye, treatment of the eye brings about gradual disappearance of the eczema.

Chronic eczema caused by the staphylococcus is actually uncommon, as compared with the frequency of infections in the outer parts of the eye. It also occurs predominantly in middle-aged women.

For 50 years it has been known that the staphylococcus produces toxic substances, but the fact received little attention for more than a quarter of a century. It was only during the past 20 years that ophthalmologists began to recognize its importance in diseases of the eye.

Such substances appear to cause trouble in two ways. First, they have a direct toxic action, and second, there seems to be an allergy to the toxin. The direct toxic action may possibly be counteracted by an antitoxin, but in some people skin tests show sensitivity to the bacterial toxins without relation to the amount of antitoxin they have in their blood. Doctor Theodore believes this type of allergy may be important in chronic eczema of the eyelids and cites other investigators whose work supports his theory. He also advances the idea that a combination of the two mechanisms brings about the painful and persistent condition.

Several methods of treatment are mentioned. Antibiotics may be useful, but some patients are sensitive to them, and moreover, staphylococci may become resistant to them. Local application gives better results than internal use. Silver nitrate solution is valuable, and ointment containing hydrocortone may also be effective, he believes. For more resistant cases, it may be necessary to give injections of toxoid or vaccine, or both, preferably made from the patient's own bacteria. Unless the underlying infection is eradicated and sensitivity to the germ eliminated, the eczema is likely to return.

AVIATION AND MILITARY OPHTHALMOLOGY

97. *Extraction of Foreign Particles of a Nonferrous Metallic Nature; Preliminary Experiments with a New Electronic Magnet.* COLONEL J. H. KING. Presented at the International Congress of Ophthalmology, New York. September 1954.

A new electronic magnet, that may make possible the removal of particles other than iron, is undergoing intensive investigation at the Army Medical Center, Washington, D. C. The research was initiated after it was found that 25 per cent of the eye injuries from foreign objects, suffered by soldiers in the Korean war, were caused by fragments of metals other than iron.

In describing this project, Colonel King pointed out that the ordinary magnet does not attract silver, brass, aluminum, copper, and various other metals, and that eyes penetrated by such substances have generally been lost despite strenuous

efforts to save them. Nonferrous metals are affected to some degree by the electromagnet, but the effect on small particles is so weak that it is of no value in saving eyes.

The most hopeful prospect was found in a modification of the electromagnet devised several years ago by Lovell. From this device, which Mr. Lovell called a "metamagnet," a special instrument, the "ocular metamagnet," has been designed.

After two years of work, during which time many models have been made, the investigators have progressed to the point of animal experimentation. Pieces of copper, aluminum, magnesium, and brass inserted in the eyes of animals have been drawn to the surface and removed. Attempts to remove particles from human eyes will not be made, however, until the instrument has been more perfected.

MEDICAL OPHTHALMOLOGY

98. *The Effectiveness of Therapeutic Procedures Dependent upon the Rationale Underlying Them.* S. HOWARD BARTLEY. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:477–487, September 1954.

This paper consists of a portrayal of the principles, explicit and implicit, that underlie various therapies in conventional medicine and osteopathy. These principles are shown to apply in contrast between optometry and ophthalmology.

Conventional thinking is shown to be disease-centered rather than patient-centered. The concepts involved in therapy are thus focused on diagnosing the disease rather than on the fundamental nature of the organism and the way that it reacts to agencies about it. The two outlooks provide contrasting ways in which to handle the patient.—*Author's abstract.*

99. *Psychosomatic Problems in Ophthalmology.* E. P. BURCH. Canad. Med. Assoc. Jour. *70*:9–17, January 1954.

Excluding persons suffering from obvious ocular diseases, at least 50 per cent of the patients consulting oculists exhibit no physical findings that could account for their complaints. Also, emotional tension may be discharged in the form of physiologic disturbances, as a spasm of the arterioles at the macula in a worried and anxious patient. The ocular symptoms in psychoneurotic disturbances may constitute only a small part of the over-all picture.

Time, effort, and patience are necessary for the diagnosis of a psychoneurosis. The examination must be thorough, the physician must be understanding and instill confidence. There are a number of psychogenic factors which may lead to a psychoneurosis. Conflicts in various spheres, frustrations often due to unreasonably high goals, and the trials of disaster and war may all breed neuroses.

In hysteria, the somatization of the psychic process occurs at the unconscious level. It often appears in those, who having been deprived of normal experiences,

have turned to fantasy. They display a vivid imagination, dramatize occurrences, are demanding and emotionally immature. The ocular findings may include amblyopia, changes in the visual fields, anesthesias, night blindness, and ptosis.

Anxiety reactions are common in practice. They often have a background of repressed emotional conflicts going back to childhood. They may be iatrogenic. The physician should take great care in all his discussions with such patients. The patient describes his symptoms in vague terms but expects absolute answers and is inflexible in his thought processes. General symptoms may include irritability, depression, palpitations, and polyurea. An examination may show a rapid pulse, cold clammy skin, and exaggerated reflexes. The patient may fear specific ocular diseases, describe head or ocular pain, or complain of photophobia, and spells of amaurosis.

The neurasthenic patient is easily fatigued, possibly due to profound emotional storms which are in themselves exhausting. He lacks drive, is irritable, and is overwhelmed by a sense of personal inadequacy. He may complain of ocular pain, spots, veils or mist before the eyes, or easy tiring of the eyes.

The obsessive ruminative-compulsion neuroses are seldom encountered by ophthalmologists. Such individuals have inflexible personalities and are incapable of enjoying the pleasures of life. They are punctilious, full of feelings of guilt and remorse, hypercritical, and intolerant. They are preoccupied with fears and worries and may exhibit ritualistic behaviorisms. These tendencies may be centered about impending blindness or specific ocular disease. There may be compulsive behavior involving the eyes.

The hypochondriac has an overriding conviction of physical disease. This psychoneurotic reaction is revealed in the patient who brings notes of his many complaints, who, on systemic inquiry, always finds further complaints, and uses many pairs of glasses.

Mixed neuroses are common and ocular reaction patterns in an individual may fit into several of the psychoneurotic groupings.

The attitude of the physician is a prerequisite to effective treatment. He must be sympathetic, thorough, unhurried, willing to listen and slow to deny the actuality of the patients' symptoms or to discount their severity.

If the ophthalmologist has sufficient time he may beneficially treat the anxiety-tension states. The psychoses and certain types of the neuroses should be referred to a specialist in psychologic medicine. These include severe hypochondriacs, obsessive ruminative-compulsion neuroses, and conversion hysterical reactions. Topic conflicts of short duration may, however, be adequately handled by the ophthalmic specialist. The treatment lies in the sympathetic unveiling of subconscious emotional factors. There are several books that might be given to the patient and could help in the therapy. Placebos and additional pairs of glasses are to be condemned, although sedatives and hypnotics may be helpful. A few interviews plus prescribed reading may fulfill the basic requirements of brief psychotherapy. 6 references.

HYGIENE, PROPHYLAXIS, OCCUPATIONAL OPHTHALMOLOGY, AND INJURIES

100. *The Importance of the A.C.A. Ratio in Industrial and School Success.* FREDERICK W. JOBE. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:521–532, October 1954.

Numerous studies have been conducted on the relationship between vision and job success. The necessity of control on age and experience has proved to be one of the greatest problems in studies of this type and has led to the search for a visual measurement which does not vary remarkably with age. The accommodative-convergence-gradient, or A. C. A. ratio, provides such a measure. The present study makes use of visual performance records submitted to the occupational research center at the Bausch & Lomb Optical company. Testing was done using the Bausch & Lomb ortho-rater which provides tests of 12 visual skills including the lateral phorias at distance and near. This permits derivation of accommodative-convergence-gradient ratios.

Experimental correlation studies of vision and job success revealed strong relationships in instances where other visual skills had no significant selectivity. A relationship between school achievement scores and the accommodative-convergence-gradient ratio is also demonstrated.—*Author's abstract.*

101. *Intra-ocular Foreign Bodies as a Surgical Emergency.* L. A. BREFFIELH. Journal of the Louisiana State Medical Society. *106*:95, March 1954.

The removal of an intra-ocular foreign body should be effected as soon as possible. When removal is delayed, the body's defenses against a foreign substance, by exudative and cellular responses, result in firm fibrous attachments to the vitreous and retina. Roentgenogram localization of the foreign body should be performed when the patient is admitted for surgery. Magnetic intra-ocular foreign bodies can be satisfactorily removed with the Lancaster hand magnet. Antibiotic medication must be administered in all cases of intra-ocular foreign bodies. Ocular irritation can be minimized and ocular tension controlled effectively by administering local cortisone.

OPHTHALMIC SOCIOLOGY

102. *A Study of Visual Performance of Mentally Retarded Children.* MORRIS KIRSCHEN. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:282–288, June 1954.

Specific information about the type and incidence of visual handicaps among the feeble minded is not easily available. In view of the paucity of such information, it was decided to investigate the visual performance of mentally retarded children at the Modesto State Hospital. The entire staff was anxious to assist in research which might shed light upon the specific visual problems of the mentally retarded child.

The author prepared for this project by setting up a battery of tests adapted

from the published works of other men who have worked with children. The work of Dr. Getman at the Yale University Institute of Child Development was helpful as was the work of Dr. Monroe Hirsch with children of grammar school age. Many of the methods used here, however, had to be curtailed because of the limited intelligence and cooperation to be expected from the group to be studied.

The tests finally decided upon were as follows: general information—name, age, I.Q.; versions—monocular and binocular, near point of convergence; cover test; ophthalmoscopy; retinoscopy; pupillary reflexes; binocularity, and visual acuity. The most significant tests were found to be the versions, cover test, and retinoscopy. The visual acuity was the most puzzling, since in a number of cases it was found that the eye closer to emmetropia had a lower acuity than the eye with greater refractive error.

The results of the survey showed about an equal distribution of males and females, the greatest number falling within the 10 to 15 year chronological age category. A large percentage had a mental age of less than 5 years with no ability to verbalize. There were many nystagmus cases uncovered and many instances of strabismus as well. Refraction disclosed more hyperopia of over 2 diopters than is found in the normal population. Finally, the author found a frequent exaggeration of symptoms both visual and general.—*Author's abstract.*

103. *The Changing Attitude Towards the Blind and the Partially Sighted.* JOSEPH I. PASCAL. Am. J. Optom. & Arch. Am. Acad. Optom. *35*:319–323, June 1954.

The old attitude that a blind man is useless is giving way to the new attitude that the blind with determination can learn to lead a useful and satisfying life.

Even more important is the new attitude towards the partially sighted. These persons have always been classified with the totally blind to the detriment of both. It is known that the partially sighted, when supplied with the newest forms of visual aids, can in the majority of instances, become fully rehabilitated. A brief historical outline is given of the origins of organized schools for partially sighted-children. The two types of schools now existing are reviewed and the superiority of the cooperative method is emphasized. A plea is made for the organization of an Institute for the Visual Rehabilitation of the Partially Sighted.—*Author's abstract.*

MISCELLANEOUS

104. *The Relationship of the Intrinsic Geometric Properties of an Object to the Apparent Shape.* THOMAS MORGAN NELSON. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:289–296, June 1954.

The author doubts the heuristic value of the concept of constancy and cites several experimental examples in which constancy has no explanatory worth. Another example is given in which a valid hypothesis, one involving an invariance of experienced object properties, may not have been confirmed because of assump-

tions inherent in the methodologic designs stemming from "object-centered' terminology. Some attention is paid to the controversy concerning "object-centered" versus "organism-centered" experimental psychologies, the author obviously favoring the latter position. The author makes an attempt to justify a reformulation of the constancy problem on the basis of a modern philosophic criterion of adequacy for scientific law, and a reformulation consistent with his experimental bias. The necessity of new terminology is stressed and several neutral terms are introduced to supplant those currently in use that, the author believes, have led to the error of "object-centered" thinking and methodology.—*Author's abstract.*

105. *Cinerama, Cinemascope and 3 D.* REUEL A. SHERMAN. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:397–410, August 1954.

Cinerama, CinemaScope, and 3 D. are manifest examples of changing visual demands that emphasize the growing importance of skillful binocular vision.

Following the opening of *Cinerama* on September 30, 1952, the motion picture industry suddenly awakened from its depression. It was a history making exhibition, organized and developed by persons from outside the motion picture industry.

The impact of *Cinerama* stimulated a search for new technics. *Bwana-Devil*, a stereo film produced on a very limited budget, was released shortly after *Cinerama* and though restricted capital did not permit adequate preparation, the film was a financial success and promoted further activity. Twentieth Century Fox also developed a panoramic process that they called CinemaScope.

Though many improvements have been made, technical problems concerning illumination, synchronization of multiple projectors and of sound and picture still exist. Each of the processes, *Cinerama*, CinemaScope, and 3 D. present visual problems for some customers.

3 D. more than any two dimensional process performs a public service for which it is often censored instead of praised. It brings to light and amplifies symptoms of inefficient binocular functions.

It is predicted that the continuing search for better motion picture entertainment will provide dynamic stereo effects combined with spectacular panoramic views.

For some persons the viewing of stereo features will help develop desirable binocular skills, but individuals with ametropia, vertical imbalances, and similar anomalies have problems that should be solved by members of the ophthalmic sciences rather than by engineers of the motion picture industry.—*Author's abstract.*

106. *Psychophysiological Aspects of Three Dimension Motion Pictures.* MONROE J. HIRSCH AND FRANK W. WEYMOUTH. Am. J. Optom. & Arch. Am. Acad. Optom. *31*:273–281, June 1954.

An earlier study by the authors of the relationship between illumination and depth perception is reviewed. The data from this study are applied to the problem of three dimension movies. It is concluded that the sensitivity of depth perception

declines if illumination levels in three dimension motion pictures are not kept up.

The problem of conflicting clues of depth, and particularly the abnormal relationships between accommodation convergence and retinal disparity, is discussed.—*Author's abstract.*

BOOK REVIEWS

Highway Safety and Driver Education. LEON BRODY, PH.D. AND HERBERT J. STACK, PH.D., New York, editors; Prentice-Hall, Inc., 1954. Pp. 447. 32 illustrations.

This volume, intended primarily for all persons interested in highway safety and driving education, actually contains very little concerning vision that would be of interest to the optometrist or the ophthalmologist. The disadvantage of tinted windshields and dark glasses for night driving are correctly stated and a paragraph on perception and driving is quite instructive. It states that, "Studies have shown that accident and violation-free drivers do not have better vision than accident repeaters and chronic violators, but apparently they 'see' the driving situation for what it is."

The authors' purpose in writing this book was to collect all available information concerning highway safety and driving education and organize it into a volume. The technical arrangement makes the material easy to find and adds to its value as a reference source. Each chapter is concluded by such topics as "Suggested Activities" and "Suggested References." It consists of 447 pages and contains 32 illustrations.—*Gerald Fonda, M.D.*

Introduction to Physiological Optics. ARMIN VON TSCHERMAK-SEYSENEGG, TRANSLATION BY PAUL BOEDER, M.D., Charles C. Thomas, Publishers, Springfield, Ill.

The purpose of this textbook is to make available to the English reading population the work of a man who has devoted his life to the teaching and research of physiological optics. It is intended for those interested in obtaining a background in this field.

For the most part the material is clearly presented. A large section of the text covers aspects of the school of ocular physiology that are little known to the English speaking people. This fact makes the translation a welcome addition to already available sources on this subject.

Many phases of the study of physiological optics are described in this book that are not to be found in any other collected source. There is for example the chapter on the "Hering" theory of color vision, supported in this book as the most acceptable theory of color vision. The sections on monocular and binocular space sense also represent much of the original material of the author.

Introduction to Physiological Optics contains 305 pages and 151 illustrations.—*Gerald Fonda, M.D.*

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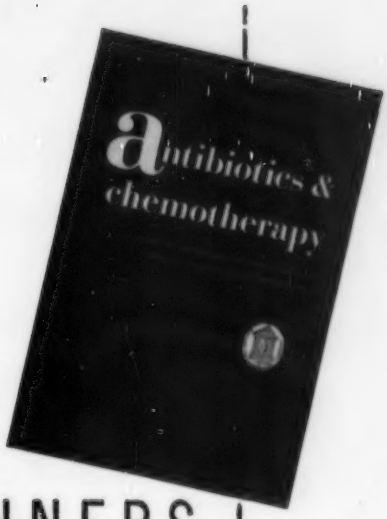

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